Initiation of Coverage
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BIOTECHNOLOGY

Equity Research

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Galapagos N.V. ADR (GLPG- $97.86)

Rating: Overweight

Price Target: $130.00

Could Filgotinib be a Best-in-Class JAK? Initiating at OW with $130 PT

REV	1Q	2Q	3Q	4Q
2017A	—	—	—	—
2018E	44.8A	57.1A	103.2A	41.5E
2019E	41.6E	41.8E	5.1E	6.2E
2020E	—	—	—	—

EPS	1Q	2Q	3Q	4Q
2017A	—	—	—	—
2018E	(0.73)A	(1.61)A	3.85A	(1.16)E
2019E	(1.14)E	(1.12)E	(0.67)E	(0.68)E
2020E	—	—	—	—

FY	2017A	2018E	2019E	2020E
REV	127.1A	246.6E	14.7E	62.0E
P/S	41.9x	21.6x	362.6x	86.0x
EPS	(2.34)A	(0.57)E	(0.21)E	(0.17)E
P/E	(41.8)x	(171.7)x	(466.0)x	(575.6)x

Summary: Initiating coverage of Galapagos at Overweight with a 12-mo. PT of $130. We think 2019 is the year that filgotinib could differentiate itself among the JAKs. Galapagos (GLPG) is a Belgian biotechnology company focused on developing therapeutics for inflammation and fibrotic conditions. Lead asset, filgotinib, is a JAK inhibitor in collaboration with Gilead (OW, A. Young) for many chronic inflammatory conditions (e.g., rheumatoid arthritis and ulcerative colitis). We think that filgotinib's potential is underappreciated and that upside in '19 could come from Ph3 readouts that will show its profile.

- **We think next-generation JAKs are the next large I&I class. We model peak unadjusted sales of $6.5B for filgotinib across various I&I conditions.** We conducted a deep-dive analysis on the emerging positioning in the JAKi landscape (see 120-page slide deck here). We think next-generation JAKs, filgotinib and upadacitinib, have significantly enhanced profiles relative to the first-generation JAKs (e.g., Xeljanz and Olumiant) and the TNFs. Among the JAK class, we think it is possible filgotinib could be best-in-class from a risk/benefit standpoint due to its highest selectivity for JAK1.
- **We are confident into upcoming Phase 3 FINCH 1 & 3 data in 1Q19.** Although expectations are high for filgotinib Phase 3 success in RA, we think that investors will assign more potential to peak sales if the data compare favorably relative to other JAKi. Key metrics we are looking at include response rates vs. competitors and infection and thrombosis rates.
- **U.S. filing timelines could drive upside if GILD/GLPG can move forward without the MANTA study.** We think the investor base case is that the male safety study, MANTA, is required for regulatory filing in the U.S. (our estimate is 2021). From speaking to both management teams, we expect that if the Ph3 risk/benefit profile is very strong, that the companies will re-engage with the FDA around a filing strategy in the US that is earlier than 2021. This would be significant since AbbVie (NC) is likely launching in late 2019/early 2020 with upadacitinib and already has a strong commercial presence with Humira.
- **Favorable risk/reward into many wild-card events such as a potential upadacitinib panel and M&A.** If Galapagos's biggest competitor asset, upadcitinib, has a panel and any incremental concerns come out, we think that would be a surprise to investors. We also think potential M&A from GLPG's partner (GILD) or others remains a swing factor.
- **Long term, we see continued innovative potential from the GLPG platform.** Galapagos uses proprietary screening capabilities to build novel oral molecules. We think filgotinib is a key piece of validation for the platform, but we think the larger value drivers from a stock perspective could emerge with wholly owned programs such as IPF, which could have data in 2020+.

Current Statistics

Market Cap ($Mil)	**$5,330**	**Shares Out (Mil) :**	**54.5**
Avg. Daily Trading Volume (3 mo.) :	**111,839**	**52 Wk. Range**	**$122.28 - $87.36**

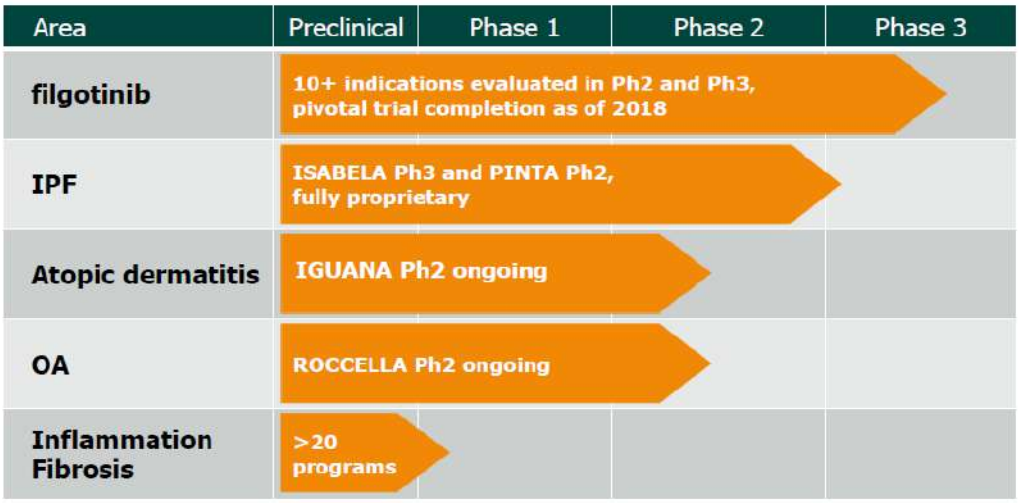
Our Galapagos thesis: We think that 2019 is the year filgotinib will differentiate itself among the JAKs. Initiating at Overweight with price target of $130.

We expect key Phase 3 data from filgotinib in studies called FINCH 1 and 3 in 1Q19. We think that these data may reveal that filgotinib has an attractive risk/benefit profile amongst the JAK inhibitors. See our separate, detailed deep dive on filgotinib and the JAK landscape here.

Galapagos is a clinical-stage biotechnology company with multiple potential blockbuster assets in Phase 3.

The company's lead asset, filgotinib, is partnered with Gilead (OW, covered by A. Young) and is in development for a variety of diseases in the inflammation and immunology (I&I) space such as rheumatoid arthritis, ulcerative colitis, and Crohn's amongst many others. Other programs in development include the wholly owned idiopathic pulmonary disease (IPF) franchise, which has entered Phase 3.

Exhibit 1: Overview of GLPG Clinical Programs



Source: Company reports

Galapagos is developing filgotinib, a JAK inhibitor that targets autoimmune conditions in a different way than traditional biologics, such as anti-TNFs (e.g., Humira and Enbrel) do.

Typical biologics work by blocking cytokine proteins that are outside of the cells and cause inflammation. JAK inhibitors prevent this inflammation by blocking the process inside the cell. There are four JAKs (JAK1, 2, 3, and TYK2) that are key components of cytokine mediated effects. The electivity for certain isoforms matters in driving a JAK inhibitor's tolerability profile. The exhibit below summarizes key safety issues to consider by isoform activity.

Filgotinib has a higher specificity for JAK1 relative to JAK2/3, which is believed to lead to a favorable tolerability profile.

Upadacitinib is described as a selective JAK1 inhibitor, but there have been various assay work and clinical data that suggest upadacitinib might also target JAK2 and JAK3 to some extent as well.

Exhibit 2: Overview of Key Safety Considerations of JAK Inhibition

Target	Safety Concerns Linked to Targeting	Drugs Targeting
JAK1	Limited	Filgotinib Upadacitinib Baricitinib Tofacitinib
JAK2	Increase in platelet count, potentially leading to elevated PE/DVT rate	Baricitinib *Upadacitinib?*
JAK3	Infection and malignancy risk	Tofacitinib *Upadacitinib?*

Source: Company reports, Cantor Fitzgerald Equity Research

In the particular case of the landscape in RA, we note that the market is crowded with many different mechanisms. However, there are only two oral products approved (the two first-generation JAK inhibitors).

Exhibit 3: Overview of Players in the Rheumatoid Arthritis Space

Key Approved Disease Modifying Agents for RA*									
Branded Name	Molecule	Company	MOA	Administration	Frequency (in RA)	Black Box	Initial US Approval	Indications+	2017 WW Sales
Humira	adalimumab	ABBV	anti-TNF	SubQ	Every other week or weekly	Serious infections and malignancy	2002	RA, JIA, PsA, AS, CD, UC, Ps, HS, UV	$18.4B
Remicade	infliximab	JNJ/MRK	anti-TNF	IV	Every eight weeks, can be increased to every 4	Serious infections and malignancy	1998	CD, UC, RA, AS, PsA, Ps	$8.2B^
Enbrel	etanercept	AMGN/PFE	anti-TNF	SubQ	Once weekly	Serious infections and malignancy	1998	RA, JIA, PsA, AS, Ps	$7.9B
Orencia	abatacept	BMY	CD80	SubQ or IV	SubQ weekly	None	2005	RA, JIA, PsA	$2.5B
Actemra	tocilizumab	ROG	IL-6	SubQ or IV	SubQ weekly or IV every 4 weeks	Serious infections	2010	RA, GCA, PJIA, SJIA, CRS	$1.9B
Simponi	golimumab	JNJ	anti-TNF	SubQ	Monthly in combination w/MTX	Serious infections and malignancy	2009	RA, PsA, UC, AS	$1.8B
Cimzia	certolizumab pegol	UCB	anti-TNF	SubQ	Every other week or every 4 weeks	Serious infections and malignancy	2008	RA, CD, PsA, AS, Ps	$1.5B
Xeljanz	tofacitinib	PFE	JAK1/3	Oral	Once or twice daily	Serious infections and malignancy	2012	RA, Ps, UC	$1.4B
Kevzara	sarilumab	REGN/SNY	IL-6	SubQ	Every other week	Serious infections	2017	RA	~$13M
Olumiant	baricitinib	LLY/INCY	JAK1/2	Oral	Once daily	Serious infections, malignancy and thrombosis	2018	RA	~$10M

*Excluding biosimilars

+ RA= rheumatoid arthritis, JIA=juvenile idiopathic arthritis, PsA=psoriatic arthritis, AS= ankylosing spondylitis, CD= Crohn's disease, UC = ulcerative colitis, Ps= plaque psoriasis, HS = hidradenitis suppurative, UV= uveitis, GCA= giant cell arteritis, PJIA = polyarticular juvenile idiopathic arthritis, SJIA= systemic juvenile idiopathic arthritis, CRS= cytokine release syndrome

^Cited 2016 sales because faced biosimilar competition in 2017

Source: Company reports

Rheumatoid Arthritis (RA) is a chronic inflammatory condition that causes joint pain, affecting more than 2 million people in the U.S. Because the disease is chronic, patients will often be treated with anti-inflammatory therapeutics for long periods of time.

There are many players in the RA space; the most commonly used mechanism is the anti-TNF inhibitor. Humira, Remicade and Enbrel are all anti-TNF inhibitors and are the cornerstone of therapy in RA. However, we think next-generation JAK inhibitors such as filgotinib can improve on the efficacy profile of the anti-TNFs and also add additional convenience (oral vs. injectibles).

Our conviction around filgotinib comes from our proprietary deep dive analysis on the JAK inhibitor space.

Our top conclusions from the report are below.

Concurrent with our Galapagos launch we are publishing a ~110-slide deep dive analysis on the JAK inhibitor space for indications related to the inflammation and immunology space. Our analysis has increased our conviction that filgotinib may have one of the most attractive profiles in the JAK inhibitor space.

- We think next-generation JAK inhibitors have enhanced efficacy and safety vs first-generation JAKs.
- We think next-generation JAK inhibitors will be blockbuster therapies, despite the fact that first-generation JAK sales have been more modest.
- We think the JAK class will sell $20B+ at peak (2026). We think filgotimib and upadacitinib may be the next analogs of Humira and Enbrel (TNF inhibitors).
- We have confidence that novel classes in many I&I therapeutics spaces can be large when efficacy and or safety is improved. A recent example of this is the psoriasis class, where novel therapies have had very successful launches despite a crowded commercial space.
- We think that thrombosis risk via JAK2 pathway will be a key safety risk to consider in this class. We will likely learn more if AbbVie's (Not Covered) upadacitinib has a panel in mid-2019. We have not seen thrombosis issues with filgotinib, though the drug's most notable issue was a preclinical finding in testes.
- We also think JAK inhibitors will have broader use than in RA alone and with potentially improved safety they could have more indications over the long term.

Cantor Call: Our Bigger Picture Thesis On Why We Like Galapagos:

We think that filgotinib is a pipeline in a product that will have greater utility than just rheumatoid arthritis. We also think the company's research platform is underappreciated when thinking about ongoing Ph3 IPF program and molecule discovery capability.

- **Idopathic pulmonary fibrosis (IPF) is a wholly owned program that has entered Phase 3 and could drive significant upside over the longer term.** We expect Ph3 data in the 2022-2023 timeframe, depending on enrollment, which means it's likely not a catalyst over the next 12 months. We don't think a significant amount of credit is in the valuation. We think that if successful, GLPG's IPF program could sell $2B+ at peak. Because this is a wholly owned program, the upside from success here is much greater. We view IPF as a high area of unmet need. The Phase 2 program was a very small dataset (20 patients), which we think is a key risk of the program.
- **In the long term, we believe strong internal R&D will drive value as new programs move into the clinic.** Galapagos has a strong track record of developing novel assets in-house. We think this scientific know-how will drive value over time and diversifies the story. We see

additional pipeline programs, for example, the proprietary Toledo program and the osteoarthritis program (partnered with Servier [private]) as additional sources of potential upside.

- **In the near term, we think filgotinib's differentiation and market opportunity is underappreciated:** We think that, as oral agents, the JAKi class has the potential to change the treatment paradigm in the I&I market ($60B+), where injectibles dominate, and we think filgotinib may have one of the best profiles.

We think upside in 2019 will be driven by filgotinib data and comparing these data to that of competitors

Although expectations are high for filgotinib Phase 3 success in RA, we think that investors may still underestimate the market potential for filgotinib. Our reasons why we think this occurs are as follows:

- **We think that investors likely view RA as an already crowded market** where each new asset has many different Phase 2 and Phase 3 studies to compare and analyze which drugs might have the most attractive profile in the real world. This is a key reason why we think our industry deep dive on the class is helpful in sifting through all of the information.

- **We think that investors are concerned about testicular findings in preclinical studies.** Filgotinib has a clean clinical profile. There was a preclinical histological finding in the testes and low sperm in two species. As a result, the FDA is requiring that filgotinib be studied in a male safety study (called MANTA) that has to be completed before a US filing to ensure there is no risk to sperm count or testicular function.

- **Galapagos/GILD are relative newcomers to the I&I space compared to AbbVie,** which is a powerhouse in the I&I space. Specifically, ABBV's Humira is the largest-selling therapeutic globally ($18B), and the company has very well established relationships with payers and rheumatologists. ABBV's upadacitinib, another next-generation JAK inhibitor, is ahead of filgotinib in time to market (US approval in 2019 for upadacitinib vs. ~2021-2022 for filgotinib).

- Upadacitinib has a large body of Phase 3 data, but GLPG's filgotinib currently has limited data in comparison. Our doctor checks indicate it is too early to tell a difference between the assets and they do not see much differentiation from the data as it stands. However, we think that as more data emerge for filgotinib over 2019 that doctors will get more bullish on the asset.

What Moves Shares in 2019, and When Does Our Thesis Play Out?

1: We are confident in clinical success for upcoming Phase 3 studies for filgotinib.

We also think that these Phase 3 FINCH studies may suggest filgotinib is best in class from a risk/benefit profile, and we think that investors will assign more potential to peak sales. Therefore, we think that the stock can trade up if these data suggest that filgotinib could have a differentiated tolerability profile. We also think if the data are robust from an efficacy standpoint and clean on safety that physicians may view the program as potentially best in class.

Although market expectations are high, we are very confident in the filgotinib Phase 3 studies (FINCH 1 & 3) working. We did a deep dive analysis in our industry report on the filgotinib clinical program, including the first Phase 3 study.

2: Filing Timelines Could Be Upside

If Gilead/Galapagos can file without the MANTA study, we think that event would be a big catalyst for GLPG shares and would lead to the drug getting to market 1-2 years earlier in the U.S.

We think the largest point of unappreciated upside is really if the Phase 3 data are clean enough to lead the regulators to be supportive of GILD/GLPG filing early. The MANTA study measures sperm count in male IBD patients and is the filgotinib study that will be needed for filing. This study has had issues enrolling, and this is a key rate-limiting step for the companies to file filgotinib. We are watching these Ph3 data closely on safety, including infections, thrombosis, and any hormonal findings in men. We think if the safety looks very clean that this increases the chance that GILD/GLPG might be able to discuss filing alternatives in the US. Since we expect ABBV to be on the market in late 2019 and is a dominant player in RA, speeding along filgotinib timelines could help US commercial competitiveness.

3: Potential Upadacitinib Panel: Favorable Risk/Reward for Galapagos

We expect an FDA advisory panel on ABBV's upadacitinib (JAK) around mid-year 2019.

We think risk/reward is positive for GLPG shares into ABBV's upadacitinib panel, where there are still some questions about the rates of thrombosis in the trials. Ultimately, we think that as long as ABBV can file the low dose of updacitinib in RA, we think upadacitinib probably comes to market with a highly competitive profile relative to the other currently approved RA drugs.

4: Potential for additional filgotinib (or JAK class) proof-of-concept readouts in more indications:

We think investors are focused on filgotinib in RA, but we note there are additional Phase 3 studies under way for filgotinib (ulcerative colitis and Crohn's) and many more Phase 2's under way as well. We expect to get Phase 3 data for filgotinib in ulcerative colitis in 2020. We also expect to get additional Phase 2 datasets over the next 1-3 years, such as in Sjogren's, cutaneous lupus, uveitis, and specific forms of Crohn's disease. We think success in additional indications will lead investors to assign more credit in potential peak sales.

5: Potential for M&A:

Gilead pays GLPG a mid-20% royalty on filgotinib and shares profits 50-50 with Galapagos in the big 5 EU and Benelux regions. We note GILD does not currently have options to anything else in the GLPG portfolio such as IPF or the newer I&I assets in early-stage development (e.g., the Toledo program).

What we know so far based on our deep dive work, gives us increased conviction that there is a large potential market opportunity for filgotinib. With data showing a favorable risk/benefit profile relative to other JAKs, we think that investors will then pay more attention to pipeline in a product, which is a key part of our thesis.

We see catalysts that investors may be overlooking that lead us to think the company may get more credit over 2019:

1) Data from the Phase 3

2) More color from the company's competitors, namely ABBV

3) Can GILD/GLPG come to an agreement with FDA on testicular toxicity?

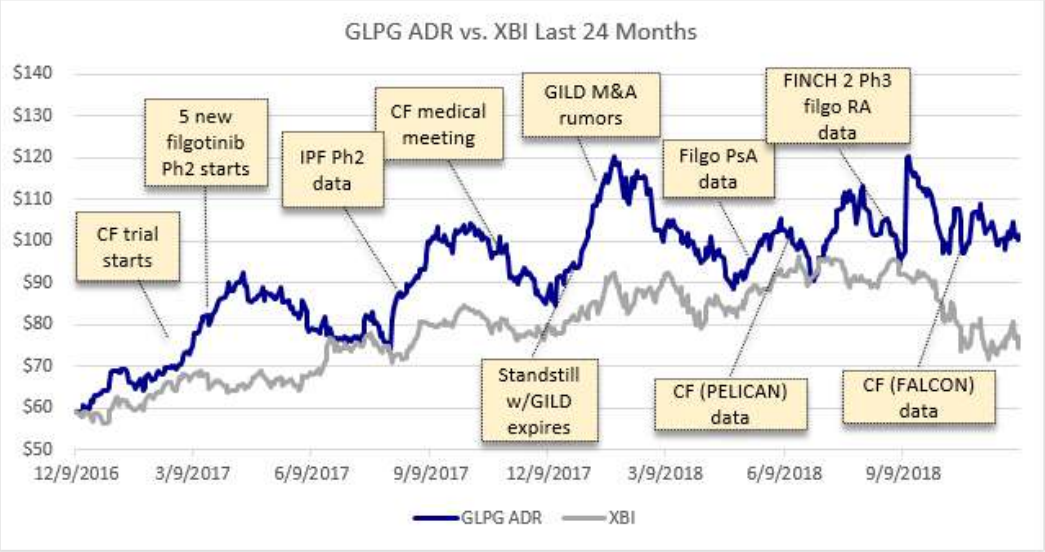
4) MANTA (male safety study) enrollment improvement

5) Potential sales in Europe

6) Phase 2 proof-of-concept studies such as Sjogren's

A key risk to shares in 2019 is any disappointing data from the Phase 3 studies in filgotinib: The FINCH 2 data came in better than our expectations from an efficacy perspective, and we think a risk from a stock perspective is if anything in the efficacy (and/or safety) profile of filgotinib seen in FINCH 2 is not replicated in the FINCH 1 and 3 studies.

Exhibit 4: GLPG vs. XBI 24 Month Performance



Source: FactSet, company reports, Cantor Fitzgerald Equity Research

Many shots on goal with strong scientific expertise also make this an attractive long-term play

<u>Thesis Point 1:</u> Lead asset filgotinib is an oral agent in development in partnership with Gilead. We forecast $6B+ in peak sales for this product in lead indications alone.

This asset is a key value driver for the company. Although we note that there are meaningful expectations already from investors for this program, we think the safety and tolerability profile as well as indication expansion could continue to pan out favorably versus competitors in the coming years.

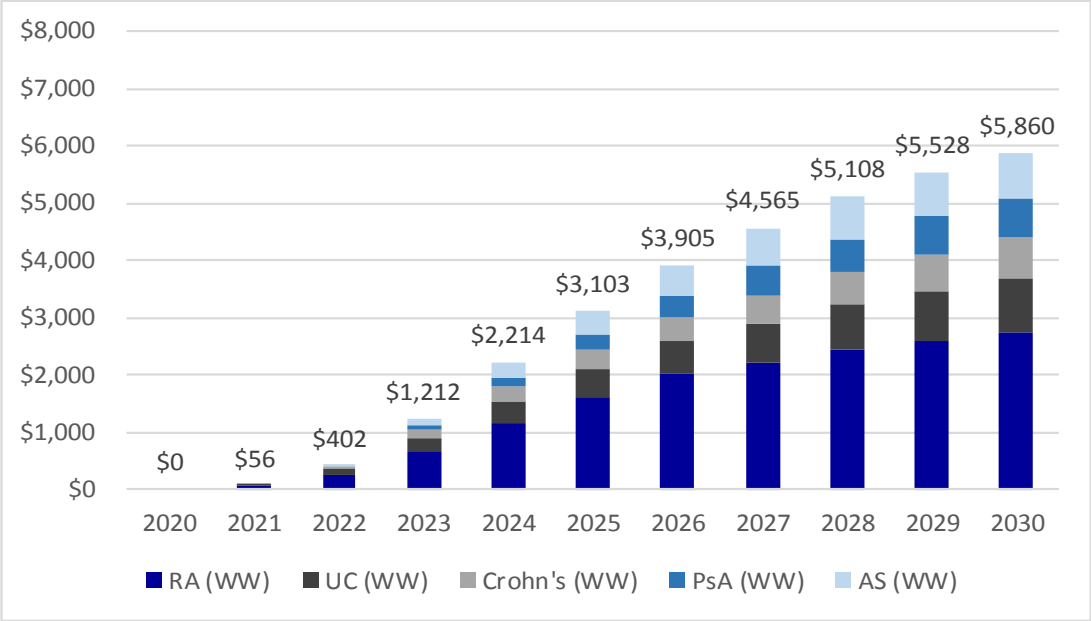
- **We think filgotinib has potential to be a key differentiated asset in the $60B+ I&I market landscape.** Filgotinib is a selective JAK1 inhibitor that has shown efficacy across many chronic inflammation and immunology (I&I) indications that we think will continue to be major therapeutic categories. Globally, the anti-TNF inhibitors alone sold $36B+ in 2017 across the variety of I&I indications in which filgotinib is being studied. We think the market for these I&I indications will continue to grow, and we see many advantages of filgotinib vs. other agents in the space. Specifically, filgotinib is an oral vs. subQ, which is the primary administration in the market, and we think filgotinib has potential for a better safety/tolerability profile vs. other agents on the market and/or in development.

Exhibit 5: Overview of Filgotinib Development Programs

Source: Company reports

- **Filgotinib is a pipeline in a product.** We think filgotinib has blockbuster potential across many large I&I indications. The asset has reported positive phase 3 data in rheumatoid arthritis (RA), but is also in Phase 3 development for ulcerative colitis and Crohn's disease and is in development for many other indications such as ankylosing spondylitis and psoriatic arthritis. With development in 10+ indications, we think this keeps GLPG a catalyst-rich story for years to come.

Exhibit 6: Cantor Unadjusted Filgotinib Sales by Indication



Source: Cantor Fitzgerald Equity Research, company reports

- **Lucrative financial arrangement for Galapagos on filgotinib.** Galapagos receives a tiered 20-30% royalty on worldwide* filgotinib sales, and commercialization and development expenses are born by Gilead. In the Big 5 EU and the Benelux countries, Galapagos will co-promote and receive half of the profits of filgotinib (but pay just 35% of the commercialization costs). This also will help Galapagos build out its own commercial presence in Europe, where it is headquartered.

- ** GLPG shares profit equally in big 5 EU and Benelux regions and receives a 20-30% royalty on sales elsewhere ex-U.S.*

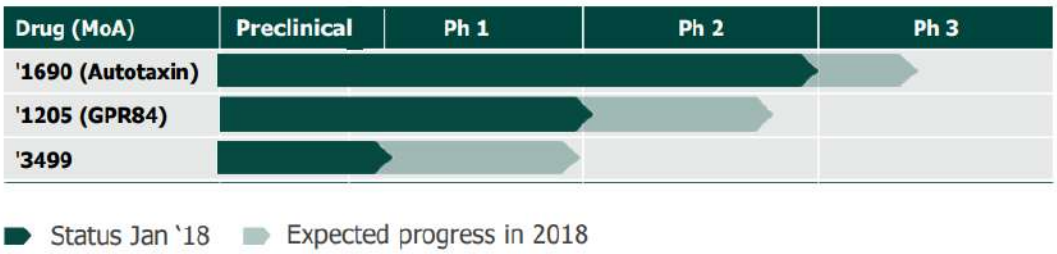
<u>Thesis Point 2:</u> We think the *wholly owned* IPF program could be a significant long-term value driver for Galapagos.

We think long-term that being wholly owned is a key upside driver, but we do not expect it to be a major stock driver in the near term. We assign limited credit to IPF in our base case, but we think the program is an interesting piece of optionality due to 1) size of the opportunity, 2) it being wholly owned, 3) the Phase 3 under way that could read out in 2022-2023 (est.), 4) relatively low expectations, and 5) supportive science and early clinical data, although limited clinical data currently.

- **We think the IPF franchise could sell $2B+ at peak if successful.** IPF is a fatal, progressive lung disease affecting nearly 100,000 patients in the U.S. There is a high unmet need for additional therapies to slow or halt the disease progression.

- **GLPG has several assets in development for IPF (idiopathic pulmonary fibrosis).** Lead asset, '1690, has entered into a large-scale Phase 3 program. GLPG1205 has entered a Phase 2 study and GLPG3499 is another asset in preclinical development. All programs apply a different mechanism of action that we think opens the potential for synergies in combination as well as many shots on goal with different assets.

Exhibit 7: Overview of GLPG IPF Franchise



Source: Company reports

- **Phase 3 data from lead asset, '1690, could be a key inflection point for GLPG in the 2022-2023 timeframe (estimate).** We think '1690 showed interesting data in a small Phase 2a study (FLORA trial). The company moved directly into a large-scale Phase 3 study. While we do not expect Phase 3 data for many years (we estimate in the 2022-2023 timeframe depending on enrollment) if this study is successful, we think it could be a major inflection point for the company with the potential to file a wholly-owned blockbuster potential drug.

- **Proof of concept data from Phase 2 PINTA study with second asset, GLPG1205, in 2020 is the next key catalyst for the IPF program in our view.** The PINTA Phase 2 study with '1205 initiated in 4Q18 and we think we could see data from this study in the 2020 timeframe (~60 patient study, 26 week primary endpoint). We have not yet seen efficacy data of '1205 in patients; if the PINTA trial is successful we think this could add increased confidence in the IPF franchise broadly with another asset that could work on its own or enhance activity of a combination.

Thesis Point 3: Other programs in development & internal R&D engine add additional shots on goal and potential for long-term value creation.

One of the reasons we like GLPG is that we think the company has a strong internal R&D engine that will produce many more shots on goal over time. Key to the company's internal R&D engine is the detailed work done around target selection. We think there is little in the stock for programs outside of filgotinib and IPF. We think success on the programs that are in or entering the clinic or success with additional pre-clinical programs that enter the clinic over time can be additional sources of long-term value.

- **We think the 'Toledo' program, a proprietary target entering the clinic for I&I conditions, is an exciting pre-clinical program:** 'Toledo' is a franchise of assets with an undisclosed target (for competitive reasons) in development for I&I diseases. The company has expressed significant excitement around this target it has identified. We think the Toledo program could move into dosing patients in 2H2019, which is when we will likely learn the target and more about the potential applications for this program.

- **Osteoarthritis asset, GLPG1972, could have Phase 2 proof-of-concept data in the 2021-2022 timeframe in a large ~850 patient study:** GLPG1972 is in Phase 2 development for osteoarthritis. GLPG has the U.S. rights, and Servier has the ex-U.S. rights to the product, which is currently enrolling a large Phase 2 trial in knee osteoarthritis (ROCCELLA trial) and was recently granted Fast Track designation.

- Atopic dermatitis (licensed to Novartis, Not Covered) and cystic fibrosis (licensed to AbbVie) are additional programs under way. We think success in either of these collaborations would drive upside to shares.

- The company has a large number of programs in pre-clinical development, and we think the company's proprietary target discovery platform could continue to deliver promising assets.

Detailed Catalyst List

Exhibit 8: Galapagos Data Readouts and Milestones

Program	What	Timing	Stock Sensitivity	Comments
Toledo	Begin Phase 1 dosing	4Q18	+	Guidance
Filgotinib - UC	Phase 3 SELECTION complete enrollment	4Q18	+	GILD guided on 3Q18 EPS call
Filgotinib - RA	FINCH 1 Phase 3 Results	1Q19	++++	GILD guided on 3Q18 EPS call Enrollment completed April 27th 2018 per ct.gov
Filgotinib - RA	FINCH 3 Phase 3 Results	1Q19	++++	GILD guided on 3Q18 EPS call Enrollment completed May 3rd 2018 per ct.gov
Filgotinib	Potentially additional data at EULAR (European Congress of Rheumatology)	June 12-15th	+++	
Filgotinib - Sjogren's	Phase 2 data	~1H 2019	++	Completed enrollment Oct 26th 2018 12 week primary endpoint
Filgotinib - Cutaneous lupus	Phase 2 data	~2019	++	Currently enrolling; 12 week primary endpoint Ct.gov lists March 2019 as primary completion date
Read to Filgotinib	ABBV upadacitinib potential panel & labeling discussion	Mid 2019	+++	
Filgotinib - RA	Potentially additional data at ACR (American College of Rheumatology)	November 8-13th	+++	
Toledo	Enter Phase 2/announce target	2H 2019	++	Cantor estimate
Filgotinib - RA	Update on regulatory path & timelines to filing in the US/ex-US	mid to 2H19 (estimated)	++++	
Filgotinib - Crohn's	Complete enrollment of Phase 3 DIVERSITY study in Crohn's	2H19	+	GILD guided on 3Q18 EPS call
Filgotinib - RA	Potential EU regulatory filing	2H19	++	Cantor estimate
Filgotinib - UC	MANTA (male safety study) enrollment update	2019 (estimated)	+++	Expect to get color on enrollment over 2019
MOR106	IGUANA Phase 2 Atopic Dermatitis results	2019 (estimated)	++	Estimated; Started dosing in May 2018, 12 week trial
TYK2	Move into Phase 1	2019	+	Per company comments
Filgotinib - Uveitis	Phase 2 data	2H 2019 (estimated)	++	Currently enrolling; 24 week primary endpoint Ct.gov lists Dec 2020 as primary completion estimate
Filgotinib - UC	Phase 3 SELECTION topline data	Mid 2020 (estimated)	++++	Induction endtpoint at week 10; Maintenance endpoint at week 58
IPF - '1205	Phase 2 PINTA results	2020 (estimated)	+++	Beginning dosing in 2H18 & 6 month primary endpoint
Filgotinib - Small bowel Crohn's	Phase 2 data	2020 (estimated)	++	Currently enrolling; 24 week primary endpoint Ct.gov lists March 2020 as primary completion date
Filgotinib - Fistulizing Crohn's	Phase 2 data	2020 (estimated)	++	Currently enrolling; 24 week primary endpoint Ct.gov lists April 2020 as primary completion date
Filgotinib - Lupus nephropathy	Phase 2 data	2020 (estimated)	++	Currently enrolling; 16 week primary endpoint Ct.gov lists July 2020 as primary completion date
Filgotinib - MANTA	Phase 2 data	2020 (estimated)	++++	Currently enrolling; 24 week primary endpoint Ct.gov lists January 2021 as primary completion date
Filgotinib - Crohn's	Phase 3 DIVERSITY results	2H20- 1H21 (estimated)	++++	Estimated based on enrollment guidance
IPF - '1690	Phase 3 ISABELA futility analysis	2020-2021 (estimated)	+++	Estimated; Beginning dosing in 2H18 & 52 week trial
OA - '1972	Phase 2 ROCCELLA results	2021-2022 (estimated)	++	Estimated; Began dosing in mid 2018
IPF - '1690	Phase 3 ISABELA Results	2021-2022 (estimated)	++++	Estimated; Beginning dosing in 2H18 & 52 week trial

Source: Cantor Fitzgerald Equity Research, company reports

Valuation and Risks: We value Galapagos shares on a probability-adjusted DCF

Exhibit 9: Detailed Valuation Assumptions by Program

PROGRAM	PROBABILITY OF SUCCESS	PEAK SALES*		PEAK TO GLPG	
Filgotinib Indications:		Unadj.	*Adj.*	Unadj.	*Adj.*
RA	100%	$2,744	*$2,744*		
Crohn's	75%	$698	*$524*		
UC	75%	$940	*$705*		
Psoriatic Arthritis	75%	$699	*$525*		
Ankylosing spondylitis	75%	$778	*$584*		
Total Filgotinib Peak Sales (unadj.)	*87%*	***$5,860***	***$5,081***	***$1,817***	***$1,580***
Cystic Fibrosis Franchise	0%	$223	*$0*	$27	$0
GLPG1690 (Idiopathic pulmonary fibrosis)	15%	$2,020	*$303*	$2,020	$303
GLPG1972 (Osteoarthritis)	0%	$1,568	*$0*	$1,002	$0
MOR106 (Atopic Dermatitis)	15%	$859	*$129*	$86	$13
TOTAL - ALL PROGRAMS		$10,530	$5,513	$4,952	***$1,896***

**We define peak as 2030*

Source: Cantor Fitzgerald Equity Research, company reports

Key Risks:

Galapagos is highly levered to the commercial potential of filgotinib. Any setback with filgotinib, could have a large impact on Galapagos's valuation. Key risks to filgotinib include:

- Efficacy seen with FINCH 2 does not hold up in the FINCH 1 & 3 trials.
- Lack of efficacy in Phase 3 trials such as ulcerative colitis, Crohn's or psoriatic arthritis.
- Safety profile from FINCH 2 does not hold up in additional studies such as FINCH 1& 3.
- Greater-than-expected competition commercially, either from additional JAK inhibitors, novel biologics, or biosimilar entrants.
- Testicular toxicity (only seen pre-clinically) is seen clinically with filgotinib.

Idiopathic pulmonary disease (IPF): A shot on a large goal with multiple studies under way in a $2B+ peak potential opportunity

Wholly owned program could make IPF a greater value driver for GLPG than filgotinib long term. We model that, if successful, the IPF franchise could sell $2B+ at peak.

In our base case we assign 15% probability of success to IPF, which is worth $16/sh to our base case. At 50% success to IPF our DCF would increase to $165/sh (30% upside from base case). At 100% success to IPF our DCF would increase to $220/sh (70% upside from base case).

Exhibit 10: Overview of GLPG IPF Portfolio

Drug	Mechanism	Stage	Next Update
GLPG1690	Autotaxin inhibitor	Phase 3 ISABELLA trials initiated 4Q18	Futility interim: ~2020-2021 (est.) Topline Ph3 data: ~2022-2023 (est.)
GLPG1205	GPR84 inhibitor	Phase 2 PINTA trial initiated 4Q18	PINTA data in 2020 (est)
GLPG3499	Undisclosed	Preclinical/Phase 1	IND enabling studies Enter clinic in 2019 (est)

Source: Company data, Cantor Fitzgerald Equity Research

Relatively limited catalysts in the next 12-18 months for the IPF program (with trials enrolling/under way) make this a longer-term value driver. However, we think it is important to consider given the potential value.

KEY TAKEAWAYS ON IPF PROGRAM:

1) We think IPF is a **very large** market opportunity.

2) Autotaxin inhibitor, GLPG1690, Phase 2 data are encouraging to us, but this is from a small phase 2 study.

3) **We think there are reasons to believe the Phase 3 with GLPG1690 could be successful** (data we estimate in the ~2022-2023 timeframe) based on the mechanism of action and data so far. A key caveat is that the Phase 3 has many differences from the Phase 2.

4) We think the **next key catalyst for the IPF program is Phase 2 proof-of-concept data for GLPG1205** in the ~2020 timeframe (PINTA study). We think favorable risk reward and success with a second asset in IPF could increase investor confidence in the IPF franchise more broadly.

5) Having multiple assets in-house with different mechanisms of action give more shots on goal with IPF. We think this is especially the case as a combination approach could enhance efficacy.

6) **This is a RISKIER Phase 3 program than most Phase 3 programs in biopharma, in our view.** 1) IPF is a difficult therapeutic space and 2) the large jump in study size to a ~1,500 patient Phase 3 from a ~20 patient Phase 2. However, from a stock perspective, we see favorable optionality here given the size of the opportunity and because, from looking at the data, we think there is a shot this works.

GALAPAGOS IPF FRANCHISE OVERVIEW: '1690 Phase 3 under way, and we expect Phase 2 proof-of-concept data for second asset, '1205, in the 2020 timeframe.

Galapagos has three disclosed assets in development for IPF. The lead asset, '1690, has recently moved into a large-scale Phase 3 program, ISABELLA (n=1500), after some encouraging, early Phase 2 data (FLORA, n=23). The second asset, '1205, is moving into a Phase 2 study, PINTA, which could have data in 2019. If successful in Phase 2, we think '1205 could help diversify the IPF program and give the company an additional shot on goal in the IPF development program.

Exhibit 11: Overview of Key Issues for the '1690 Program

Overview of Debates Around IPF Program	
Key Positives	IPF is a very large market opportunity with limited competitors
	Promising Phase 2 (FLORA) data w/ '1690 showing a clinically meaningful difference on forced vital capacity (phase 3 endpoint)
	Autotaxin ('1690) is a recognized mechanism of action
	Multiple shots on goal w/several clinical assets
	Encouraging pre-clinical data for '1205 suggesting greater activity than '1690
Key Negatives/ Risks	Many agents have failed in this space in the past. Historically, IPF has been a very difficult disease for drug development
	FLORA data were in a small dataset of ~23 patients vs. Ph3 with 1500
	Many changes from Ph2 to Ph3 (monotherapy vs. combination, study size, study length, dose levels)

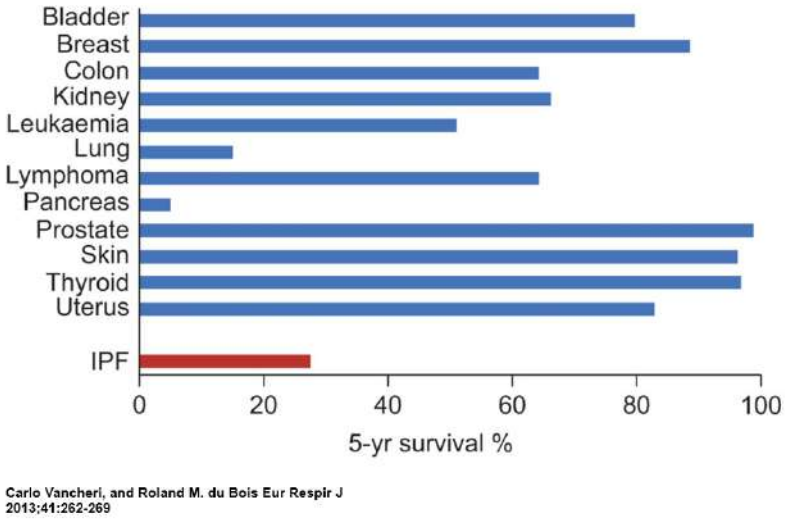
Source: Company data, Cantor Fitzgerald Equity Research

Disease Background:

IPF is a high unmet-need, fatal disease that has a prevalence of over 100k patients in the U.S.: IPF is a progressive lung disease where fibrosis worsens over time and ultimately leads to respiratory failure and death. There are up to as many as ~20k deaths per year in the U.S. due to IPF, and there are currently limited treatments available to slow the progression of the disease.

Currently the survival rate is more severe for IPF than many cancers, demonstrating the degree of unmet need in IPF. Existing treatments (e.g., nintedanib and pirfenidone) slow progression, but do not halt or stabilize the progression. In addition, current treatments come with significant tolerability issues and have very low adherence rates, further limiting their activity.

Exhibit 12: Five-Year Survival Rate for IPF vs. Various Cancers



Source: European Respiratory Society, 2013

Large and growing prevalence: In the U.S., the prevalence is estimated at ~100k patients, with an incidence of ~30-40k new patient diagnoses per year. We think if the survival were to increase, the prevalence would also grow significantly given the high incidence rate.

Standard of care has significant room to improve on both efficacy and tolerability:

- Standard of care is treatment with pirfenidone (Esbriet; marketed by Roche, Not Covered) or nintedanib (Ofev; marketed by Boehringer Ingelheim)
- Both agents slow the progression of the disease, but come with tolerability and safety issues
- In addition, while these agents slow the progression of the disease, they do not halt or reverse the disease progression

GLPG1690: Autotaxin inhibitor and company's lead asset for IPF

Large-scale Phase 3 under way; we do not expect data until the 2022-2023 timeframe, but we think there is highly favorable risk/reward into this update given the significant value potential.

'1690 has seen interesting Phase 2 data, albeit in small numbers, and has moved into a large Phase 3. GLPG1690 is an autotaxin inhibitor. Autotaxin is an enzyme that generates most lysophosphatidic acid (LPA), which is a molecule believed to be a key driver of pro-fibrotic activity in the lung. In a small Phase 2, patients dosed with GLPG1690 had a meaningfully slower rate of disease progression vs. placebo. Given the severity of disease, if these results were to hold up in larger trials, this would be a highly meaningful effect for patients and the community. Notably, LPA levels, the mechanism by which autotaxin is intended to work, were lowered in response to GLPG1690 dosing, suggesting the hypothesis could be panning out.

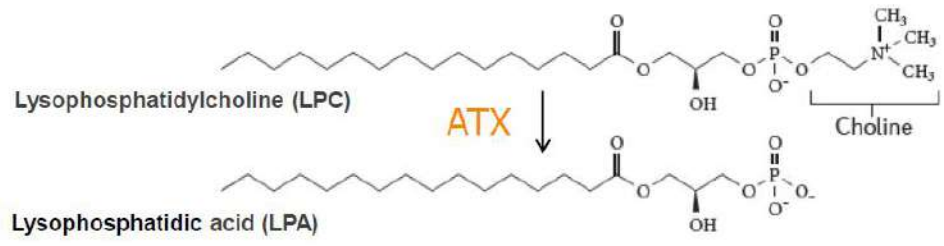
Exhibit 13: Pros and Cons on the '1690 Program in IPF

Key Reasons in Support of '1690 in IPF
FCV benefits vs. placebo in Ph2a: Phase 2 showed clinically meaningful difference at 12 weeks on FVC (forced vital capacity) the well-accepted endpoint for Phase 3 studies in the space
Successful target engagement: LPA levels (the mechanism autotaxin is intended to work by) decreased with '1690 dosing
Autotaxin target validation from BMS asset. BMS's asset, BMS-986020, which is an LPA1 inhibitor (downstream of autotaxin) helped validate the autotaxin target as it showed statistically significant benefits on FVC vs. placebo (but had asset-specific toxicity issues) from this pathway
Key Questions/Drawbacks on the '1690 Program
Phase 2a data was in a small number of patients (n=23)
In Phase 2a FVC varied over time
The FVC difference in Phase 2a was only statistically significant at week 8, but not stat. sig at the other time points.
Many differences between the Phase 2a and Phase 3 trial designs introduce more variables: - Studied in combination w/standard of care in ph3 vs. monotherapy in ph2a - Endpoint at 52 weeks in Ph3 vs. 12 weeks in ph2a

Source: Company data, Cantor Fitzgerald Equity Research

GLPG1690 is an autotaxin inhibitor, which reduces LPA evels. Autotaxin is one of the main enzymes that produces lysophosphatidic acid. LPA has been linked to many pro-fibrotic pathways in the lung, and thus reducing LPA is believed to modulate the fibrosis in the lung that occurs in IPF.

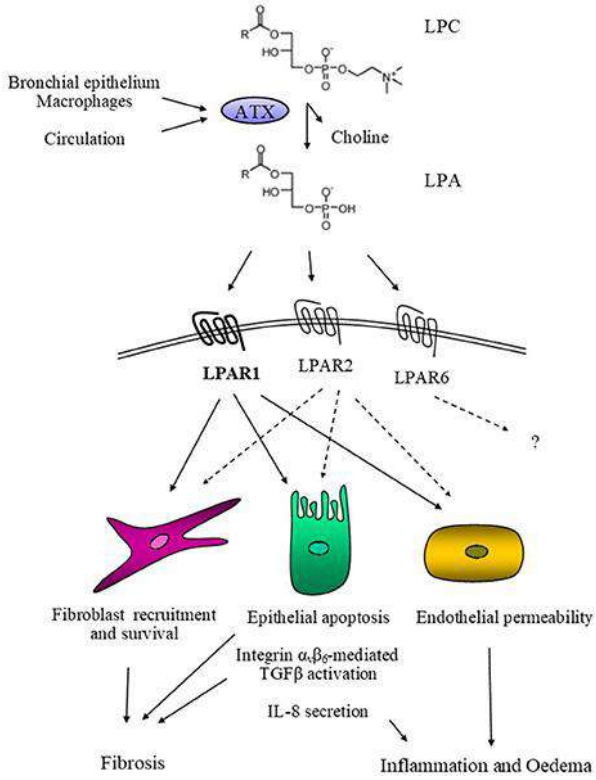
Exhibit 14: Overview of Autotaxin (ATX) Mechanism of Action



as a lungs.

Source: Company data

Exhibit 15: Lysophospholipid Signaling



Source: Ninou et al 2018

In the Phase 2a (FLORA study) '1690 showed a difference on FVC, the key endpoint in IPF trials. Forced vital capacity (FVC) is the key measure of efficacy in IPF. It is a measure of lung capacity and lung function by measuring how much air a person can exhale in a breath. This is the standard primary endpoint for studies in IPF. Approved agents, Ofev and Esbriet, have demonstrated a slowing in FVC declines relative to placebo. However, no agent has been able to improve FVC or halt the declines in FVC. In Phase 2a, although in small numbers, GLPG1690, was able to increase FVC in some patients and showed a meaningful difference vs. placebo patients.

Exhibit 16: Forced Vital Capacity (FVC) Changes in FLORA Ph2a Study

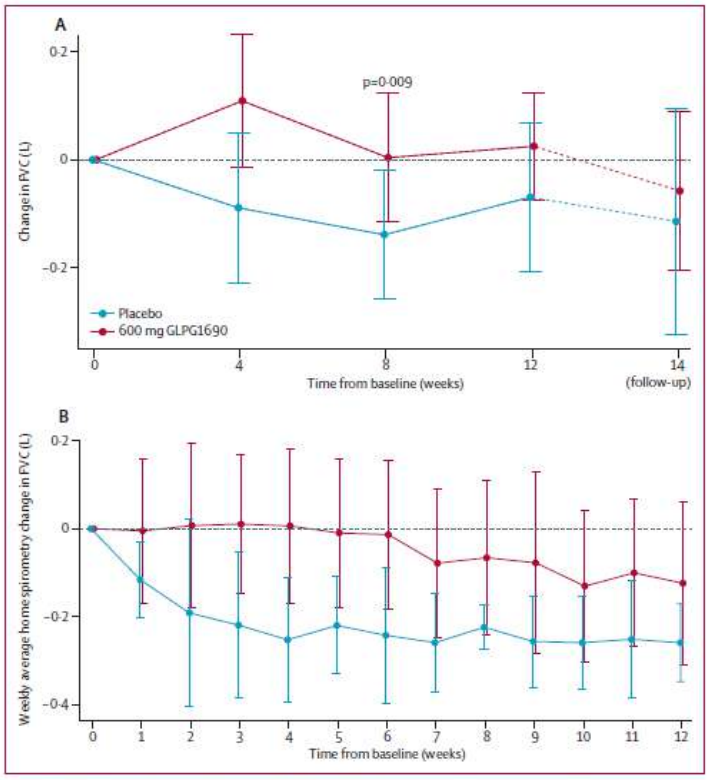


Figure 3: **Mean (95% CI) changes in FVC from baseline in the placebo and GLPG1690 groups in the intention-to-treat population**
(A) Spirometry results from study centre visits; placebo group n=6 and GLPG1690 group n=17; changes were non-significant at weeks 4 (p=0·13), 12 (p=0·3), and 14 (p=0·06). (B) Spirometry results from measurements at home; placebo group n=6 and GLPG1690 group n=16. FVC=forced vital capacity.

Source: Maher et al, Lancet 2018.

The change in FVC is the primary endpoint in the Phase 3 study (ISABELLA), which is a measure in which '1690 showed a difference vs. placebo in phase 2a.

If similar differences are seen in Phase 3 between the treated arm and the placebo on FVC, this would very likely be enough to be statistically significant, in our view. In Phase 2a, a 95mL difference in FVC was seen between placebo and '1690-dosed patients at week 12. The Phase 3 study is powered to show an 80mL difference in FVC at week 52. This is powered to show a slightly lower FVC difference than was seen in Ph2a. We note, however, that with a much larger sample size we would expect the potential magnitude of effect seen to go down. The company believes that the difference vs. placebo will widen over time and so the longer treatment period (52 vs. 12 weeks) is a benefit and could increase the activity.

Exhibit 18: Mean Change in FVC (Forced Vital Capacity) in Phase 2a and Phase 3 FVC Powering

	Mean Change in FVC	Range
GLPG1690	+25mL	-25 to +124
Placebo	-70mL	-208 to +68

Source: Company Data

	Difference vs. Placebo	Endpoint Measured At:
Phase 2 Showed:	95mL difference	12 weeks
Phase 3 **Powered** for:	80mL difference	52 weeks

Source: Company Data

Exhibit 17: GLPG1690 Phase 3 Design

ISABELA 1 &2	
n	1500 (combined)
Primary Endpoint	Rate of decline of FVC (in mL) at week 52
Secondary Endpoints	Respiratory hospitalizations Mortality
Study Length	52 weeks
Regimen	GLPG1690 or Placebo Two doses being studied
Background Therapy	On top of existing standard of care (e.g. Esbriet or Ofev)
Study Start	2H 2018
Powering	90% power to detect an 80ml difference

Source: Company data, Cantor Fitzgerald Equity Research

A key change in the Phase 3 study is that it is looking at '1690 in combination with standard of care (Ofev or Esbriet), whereas the Phase 2a (FLORA) study looked at '1690 as monotherapy. We do not know the exact clinical impact of studying '1690 in combination with other agents. We assume this was a design characteristic that was made in order to satisfy regulatory requirements and to help encourage enrollment. Pre-clinical work has shown that different mechanisms of action (e.g., with Esbriet and Ofev) can lead to increased efficacy when used in combination, but the use of these combinations in practice is limited by the toxicity of these assets. GLPG1690 so far has not been associated with toxicity/adverse effects relative to placebo, so this could be an advantage of this asset.

The Phase 3 is also studying a longer treatment period (52 weeks vs. 12 weeks for Ph2) and is in a much larger population (~1500 vs. 23, respectively). This is a very sizeable jump in size of study, but the company wanted to pursue the program as quickly as possible and baked in an interim for futility after 25% of patients have completed the study.

Exhibit 19: Key Differences from Ph2 to Ph3 for '1690 in IPF

	Phase 2	Phase 3
Regimen	Monotherapy	Combination Therapy w/Standard of Care
Dose Level	600mg once daily	Also studying a lower dose as well as ph2 dose
Geography	2 sites in UK 6 sites in Ukraine	Mostly EU and US sites
Patient Number	23	1500
Timeframe	12 weeks	52 weeks

Source: Company data, Cantor Fitzgerald Equity Research

Assuming the Phase 3 takes 24-36 months to enroll the ~1,500 patients, we think we could see data in the 2022-2023 timeframe. Enrollment timelines are a large swing factor in terms of when there could be data. If the trial enrolls in 12 months (e.g., completes enrolling in late 2019/early 2020), we could potentially see data in 1H 2021, which would be a major milestone for the company. However, we have limited visibility on how long it will take the trial to enroll, but we do expect updates on how enrollment is going over the course of 2019.

For context, the Ofev (nintendanib) Phase 3 trial took 16 months to enroll ~1,000 patients (last drug approved for IPF) in 2011/2012, but this was a smaller study vs. GLPG's 1,500. However, in the ISABELA trial '1690 is also being studied in combination with standard of care, which could help encourage enrollment.

Exhibit 20: Overview of Estimated '1690 Timelines

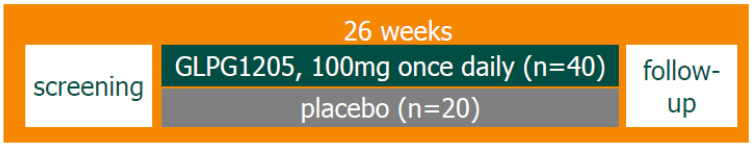
What	When	Comments
Study Start	4Q 2018	Company guidance
Enrollment Completion	~2021 -2022	Estimation - assume it takes 24-36 months
Phase 3 futility analysis	~2020-2021	Estimation - company says will do a futility analysis when ~25% of patients are 1 year into treatment
Phase 3 efficacy readout	~2022 -2023	Estimation - primary is at 52 weeks

Source: Company data, Cantor Fitzgerald Equity Research

A futility analysis will be conducted when ~25% of patients have completed the Phase 3 study, which could be a milestone. While typically passing a futility analysis is not much of a catalyst, given the small amount of clinical data on this asset, we think the futility could be a catalyst for the asset. Specifically, the futility analysis, which could come in the ~2020-2021 timeframe (depending on enrollment) could signal that the drug is having some sort of effect on the disease.

In the meantime, we think the next catalyst that could lead investors to assign more credit to the IPF franchise could be the PINTA ('1205) Phase 2 proof-of-concept study. This is with the company's second asset for IPF, '1205, that has a different mechanism of action. The trial started in late 2018 and, depending on enrollment, we think we could see data in 2020 (26-week primary endpoint). We note that this study will also be larger vs. the FLORA study with a larger number of patients.

Favorable risk reward into this data: We think success in the PINTA study will increase investors' success assigned to the IPF platform broadly. However, because '1205 is a separate mechanism of action vs. '1690, if the trial is not successful we do not think there will be a meaningful read to make to the '1690 ISABELA Phase 3 program under way.



- 60 IPF patients on local standard of care
- Primary endpoint: forced vital capacity (FVC) at 26 weeks
- Secondary: safety, tolerability, broad range of measurements, incl. FRI
- Recruitment in 10 countries in Europe, North Africa, & Middle East

Source: Company data

Exhibit 21: PINTA Phase 2 Trial Design

GLPG1205 is a GPR84 inhibitor. GPR84 is a relatively more novel target than autotaxin in its application to IPF. GPR84 is associated with metabolic inflammation and is believed to be linked to the pathway between obesity and diabetes. Recent studies, such as the work done by Promedic, have demonstrated a link between GPR84 (and GPR40) and organ fibrosis.

Competitive landscape: Fibrogen (Not Covered), Biogen (OW, covered by A. Young) and Promedior (Not Covered) also have programs in mid- to late-stage clinical development for IPF. Our general take, however, is that this is a very large population with few available therapies, and so we do not view this as a highly competitive space given the lack of effective treatments so far. In addition, we think combination therapy longer term with multiple different mechanisms of action could make sense from a clinical perspective.

Exhibit 22: Key Players in Development in the IPF Space

Asset	Company	MOA	Stage	Comments
GLPG1690	Galapagos	Autotaxin	Phase 3	Initiating Ph3 in 4Q18
Pamrevlumab	Fibrogen	Connective Tissue Growth Factor (CTGF)	Phase 2 (entering Ph3)	Awaiting minutes from end of Ph2 meeting w/FDA
BG00011	Biogen	Integrin alpha-V beta-6 (modulates TGF-beta)	Phase 2b	Began dosing Ph2b in 3Q18
PRM-151	Bristol/Promedior[1]	Pentraxin-2	Phase 2	Ph2 completed in May 2018
CC-90001	Celgene	c-Jun N-terminal kinase (JNK)	Phase 2	
KD025	Kadmon	ROCK2 (Rho-associated coiled-coiled kinase 2)	Phase 2	
PBI-4050	Prometic Life Sciences	GPR40 and GPR84	Phase 2	
TD-139	Bristol/Galecto[2]	Galectin-3 (inhaled)	Phase 1/2	
GLPG	Galapagos	GPR84	Phase 2	
GLPG	Galapagos	Undisclosed	Preclinical	
PLN-74809	Pliant Therapeutics	Integrin alpha-V beta-6 and alpha-V beta-1 dual inhibitor	Preclinical	

1 Bristol has the exclusive right to acquire Promedior

2 Bristol has the exclusive right to acquire Galecto

Source: Company data, Cantor Fitzgerald Equity Research

Management Biographies

From the Galapagos company website:

Onno van de Stolpe - Chief Executive Officer

Onno van de Stolpe founded the company in 1999 and has served as the Chief Executive Officer and a member of the board of directors from 1999 to the present. From 1998 to 1999, he was the Managing Director of Genomics at IntroGene B.V. (later Crucell N.V., which was acquired by Johnson & Johnson Services, Inc. in 2011). Prior to joining IntroGene in 1998, he was Managing Director of Molecular Probes Europe B.V. He established this European headquarters after joining Molecular Probes, Inc. in the United States. Previously, he worked for The Netherlands Foreign Investment Agency in California, where he was responsible for recruiting biotechnology and medical device companies to locate in the Netherlands. Mr. Van de Stolpe started his career as Manager of Business Development at MOGEN International N.V. in Leiden. He received an MSc degree from Wageningen University. Mr. Van de Stolpe currently also serves as a member of the supervisory board of the Stichting Institute for Human Organ and Disease Model Technologies and previously served as a member of the board of directors of DCPrime B.V.

Piet Wigerinck, Ph.D. - Chief Scientific Officer

Piet Wigerinck joined Galapagos in April 2008 from Tibotec-Virco Comm. VA (a subsidiary of Johnson & Johnson), where he was VP Drug Discovery, Early Development and CM&C, and a member of the Management Board. He started his professional career as a medicinal chemist at Janssen Research Foundation in 1992. He then joined Tibotec Group NV in 1998, where, under his leadership, TMC114 (Prezista™) and TMC435 (Olysio™) were selected and moved forward into clinical trials. Dr. Wigerinck also played a key role in Tibotec's expansion into novel diseases such as Hepatitis C and advanced several compounds into Phase 1 and Phase 2 clinical trials. He brings over 25 years of research and development experience from both large pharmaceutical companies and biotechnology companies to Galapagos. Dr. Wigerinck holds a Ph.D. from the K.U. Leuven and is inventor on more than 25 patent applications.

Bart Filius, MBA - Chief Operating Officer & Chief Financial Officer

Since September 2017, Bart Filius is the Chief Operating Officer, as well as the Chief Financial Officer. He joined Galapagos in December 2014 as CFO. Prior to that, Mr. Filius worked over 13 years at Sanofi S.A., where he was Chief Financial Officer of Sanofi Europe during the last three years and was instrumental in transforming the Sanofi European organization to be well-positioned beyond the patent cliff. Earlier at Sanofi, Mr. Filius was CFO and Country Manager of Sanofi in the Netherlands. Before that, he was Vice President for Mergers & Acquisitions, during which time Mr. Filius led and completed the divestiture of various franchises. Prior to joining Sanofi, Mr. Filius was a strategy consultant at Arthur D. Little. Mr. Filius is a Dutch national and has an MBA degree from INSEAD and a bachelor's degree in business from Nyenrode University.

Andre Hoekema, Ph.D. - Chief Business Officer

Andre Hoekema joined Galapagos in March 2005 from Invitrogen Corporation, where he was Managing Director of Corporate Development Europe. He brings 20 years of biotech experience from positions at Molecular Probes Europe B.V. (Managing Director), Crucell N.V. (Director of Business Development), DSM Life Sciences N.V. and Syngenta MOGEN B.V. (Research and Project Management) and Genentech, Inc. (R&D). Dr. Hoekema has a Ph.D. degree from Leiden University and is the inventor of over 20 series of patent applications, resulting in 15 patents issued in the U.S. Dr. Hoekema currently also serves as a member of the supervisory board of Mimetas B.V.

Walid Abi-Saab, M.D. - Chief Medical Officer

Walid Abi-Saab joined Galapagos as Chief Medical Officer in March 2017. Dr. Abi-Saab drives the overall medical strategy of the company and is responsible for late-stage clinical development and operations, medical and regulatory affairs, and safety. Previously, Dr. Abi-Saab worked at Shire Pharmaceuticals where he held various clinical development leadership roles, most recently as Group Vice President, Global Clinical Development - Therapeutic Area Head, Gastro-intestinal, Endocrinology and Metabolism. Prior to that, he led clinical development activities at Novartis, Abbott Laboratories and Pfizer, addressing a wide range of therapeutic areas and leading teams throughout the clinical development process. Under his leadership, more than 30 molecules have advanced through clinical development leading to several approvals in the US, EU and Canada. Prior to his pharma roles, Dr. Abi-Saab was Assistant Professor of Psychiatry and Neurosurgery at Yale University Medical School, where he headed their Schizophrenia Research at the Clinical Neuroscience Research Unit and the Neurosurgery Epilepsy Microdialysis Research Program. Dr. Abi-Saab holds an M.D. degree from Université Saint Joseph in Beirut, Lebanon.