BRYAN, GARNIER & CO

2nd October 2018

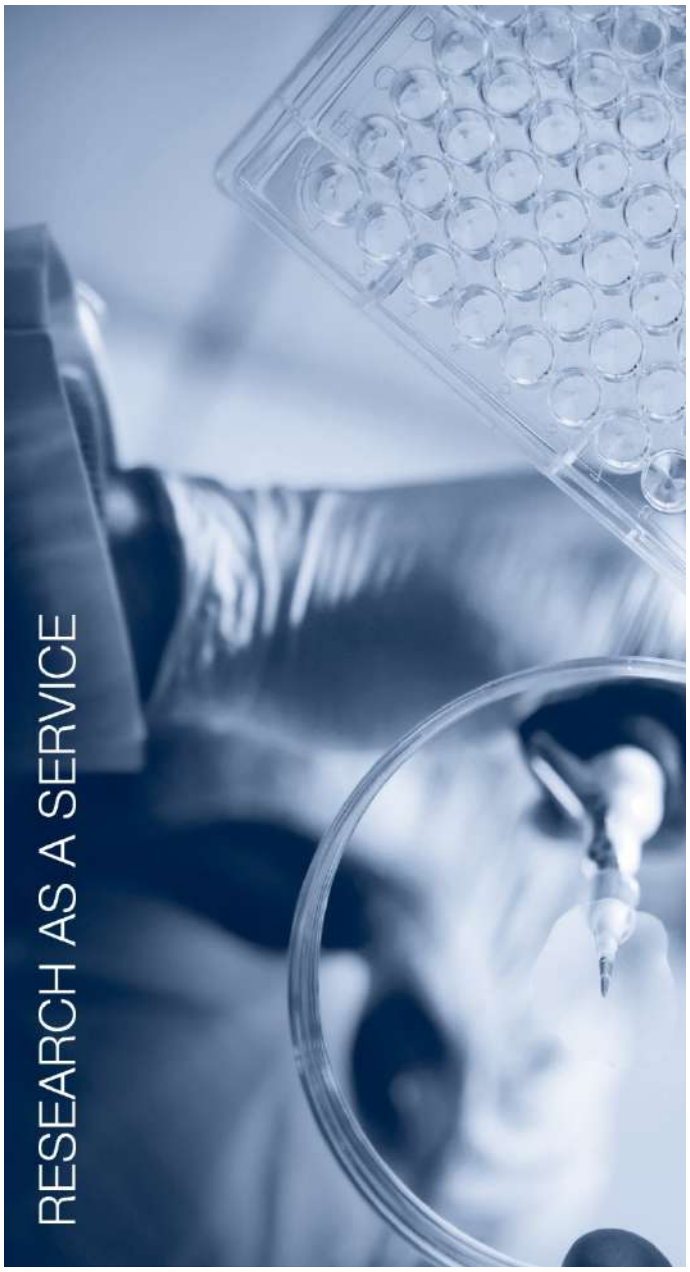
Galapagos

BUY

Fair Value	**EUR120**
Share price	EUR96.26
Bloomberg / Reuters	GLPG BB/GLPG.BR

Healthcare | **Biotech**

Slashed Price for a De-risked Blockbuster and Rich Pipeline!



Following the readout of the FINCH-2 trial for filgotinib in rheumatoid arthritis as well as recent promising phase II results in ankylosing spondylitis, we consider the blockbuster status of filgotinib de-risked and estimate non-risk adjusted peak sales at EUR6bn. Additional read-outs for filgotinib, partnered with Gilead, should drive significant value creation during 2019.

The current share price seems almost entirely justified by filgotinib (BGe EUR86/share), and does not include GLPG1690 in idiopathic pulmonary fibrosis, MOR106 in atopic dermatitis and GLPG1972 in osteoarthritis. While GLPG1690 (phase III trial ongoing), could prove to be the first molecule to halt the progression of IPF, MOR106 was recently in-licensed by Novartis. Although more risky, proprietary GLPG1972 could reshuffle the cards in a high unmet need.

Lastly, we see very limited downside risk ahead of the interim results from FALCON, evaluating GLPG/ABBV's first triple combo in cystic fibrosis. We have low expectations on the outcome of this interim as GLPG2737 is unlikely to have reached maximum exposure levels. This, alongside a partnership currently being reviewed, has prompted us not to include this programme in our valuation.

We value Galapagos at EUR120 per share, pointing to upside of around 25% relative to the current share price. The newsflow concerning filgotinib over 6-12 months only could drive our FV to EUR135/share.

Hugo Solvet |33(0) 1 56 68 75 57| hsolvet@bryangarnier.com

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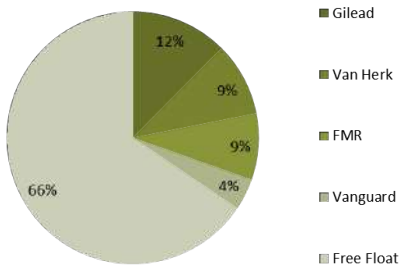
Galapagos

BUY

Fair Value	**EUR120 (+25%)**
Share price	**EUR96.26**
Market Cap.	**EUR5,227m**

Shareholders



Income Statement (EURm)	2016	2017	2018e	2019e	2020e	2021e	2022e
Revenues	152	156	178	178	186	161	276
Change (%)	150%	3%	14%	0%	4%	-13%	71%
EBIT	-11	-90	-100	-149	-135	-104	-112
Change (%)	-87%	681%	11%	49%	-9%	-23%	8%
Financial results	66	-26	4	4	3	3	2
Pre-Tax profits	54	-116	-96	-145	-132	-101	-110
Tax	0	0	0	0	0	0	0
Net profit	54	-116	-96	-145	-132	-101	-110
Restated net profit	54	-116	-96	-145	-132	-101	-110
Change (%)	ns	-314%	-16.7%	-50.1%	-9.0%	-23.3%	-8.6%
Cash Flow Statement (EURm)							
Operating cash flows	5	-69	-95	-144	-130	-99	-107
Change in working capital	-269	314	-11	20	-75	14	-8
Capex, net	-4	-5	-5	-5	-5	-5	-5
Financial investments, net	396	353	300	0	0	0	0
Net debt	-970	-1 148	-1 281	-1 045	-898	-768	-637
Balance Sheet (EURm)							
Tangible fixed assets	15	17	18	20	22	23	25
Intangibles assets	1	2	4	5	7	8	10
Cash & equivalents	973	1 151	1 285	1 050	903	773	643
current assets	34	46	46	46	46	46	46
Other assets	60	69	67	67	67	67	67
Total assets	1 083	1 286	1 421	1 189	1 045	919	791
L & ST Debt	107	175	156	80	78	71	56
Others liabilities	217	99	47	37	26	7	5
Shareholders' funds	759	1 012	1 217	1 073	941	840	730
Total Liabilities	1 083	1 286	1 421	1 189	1 045	919	791
Financial Ratios							
Operating margin	-7.6%	-57.6%	-56.1%	-83.4%	-72.5%	-64.4%	-40.7%
Net margin	35.6%	-74.2%	-54.1%	-81.2%	-70.8%	-62.6%	-39.8%
Pay out ratio	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Number of shares, diluted	47	50	54	54	54	54	54
Data per Share (USD)							
EPS	1.14	-2.33	-1.79	-2.68	-2.44	-1.87	-2.03
Restated EPS	1.14	-2.33	-1.79	-2.68	-2.44	-1.87	-2.03
% change	ns	(304%)	(23%)	(50%)	(9%)	(23%)	(9%)
Operating cash flows	0.11	-1.39	-1.77	-2.67	-2.41	-1.84	-1.99
Net dividend	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Hugo Solvet

33(0) 1 56 68 75 57

hsolvet@bryangarnier.com

TEAM :

E Le Berrigaud	eleberrigaud@bryangarnier.com
J.J. Le Fur	jjlefur@bryangarnier.com
G Waanders	Gwaanders@bryangarnier.com
V Floc'h	vfloc@bryangarnier.com

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EXECUTIVE SUMMARY

Following the readout of the FINCH-2 trial for filgotinib in rheumatoid arthritis, recent promising phase II results in ankylosing spondylitis and in psoriatic arthritis, we consider the blockbuster status of filgotinib de-risked and estimate non-risk adjusted peak sales at EUR6bn. Additional read-outs for filgotinib should drive significant value creation during 2019. In H1 2019, we would expect the FINCH-1 and FINCH-3 trial to readout. Alongside the initial readout of the MANTA trial investigating testicular toxicity in ulcerative colitis patients, Gilead should have enough data to file for approval in H2 2019 (BGe). In late 2019/early 2020, two additional phase III trial in ulcerative colitis and Crohn's disease should readout.

The current share price is almost fully supported by filgotinib (BGe EUR86/share), and does not include: 1/ GLPG1690 in idiopathic pulmonary fibrosis which could prove to be the first molecule to halt the progression of the disease. GLPG1690 benefits from a strong support from patients' advocacy groups and the FDA which authorised the ISABELA phase III program to enrol over >1,500 patients on the back of a 23-patient phase IIa. 2/ MOR106, investigated in atopic dermatitis and recently in-licensed by Novartis in a >EUR1bn deal. 3/ GLPG1972, a first-in-class ADAMTS-5 which could reshuffle the cards in osteoarthritis, a high unmet medical need and on which Galapagos retains full US rights.

Lastly, we see very limited downside risk ahead of the interim results from FALCON, evaluating Galapagos and AbbVie's first triple combo in cystic fibrosis patients. We have low expectations on the outcome of this interim as GLPG2737 is unlikely to have reached maximum exposure levels at week 2. This, alongside a partnership currently being reviewed, has prompted us not to include the fibrosis programme in our valuation.

We value Galapagos at EUR120 per share, c.25% upside potential relative to the current share price. Newsflow concerning filgotinib over 6-12 months only could drive our FV to EUR135/share. A settlement with AbbVie on the cystic fibrosis programme by 2018YE as well as additional details on the early stage pipeline to be presented at the R&D day later this month (Oct. 25th) should further strengthen the long term growth story of GLPG

Suite aux résultats des études FINCH-2 dans la polyarthrite rhumatoïde, de phase II dans la spondylarthrite ankylosante et l'arthrite psoriatique, nous considérons le potentiel de blockbuster de filgotinib dé-risqué et estimons un pic de ventes de EUR6bn. Un newsflow dense devrait supporter la création de valeur en 2019. 1/ Au S1 2019, nous attendons les résultats des études FINCH-1 et FINCH-3. Adossés aux résultats initiaux de l'étude MANTA investiguant la toxicité testiculaire de filgotinib dans la colite ulcéreuse, Gilead devrait pourvoir soumettre filgotinib aux autorités règlementaires dans le courant du S2 2019. 2/ A horizon fin 2019/début 2020, deux études de phase III dans la colite ulcéreuse et la maladie de Crohn devraient dévoiler leurs résultats.

Nous estimons que le cours actuel du titre est presque entièrement supporté par le filgotinib (EUR86/titre) et ne prend pas en compte : 1/ GLPG1690 dans la fibrose pulmonaire idiopathique. GLPG1690 bénéficie d'un fort soutien des associations de patients et de la FDA. Cette dernière ayant validé le programme de phase III ISABELA chez plus de 1500 patients sur la base de résultats d'une phase IIa conduite chez 23 patients. 2/ MOR106, étudié dans la dermatite atopique et récemment licencié à Novartis pour plus de EUR1bn. 3/ GLPG1972 qui pourrait néanmoins rebattre les cartes dans l'ostéoarthrite, un besoin médical non satisfait dans lequel la société retient les droits US du produit.

Enfin, nous voyons un risque de baisse limité à l'aube des résultats intérimaires de l'étude FALCON évaluant la première combinaison triple de GLPG/ABBV dans la mucoviscidose. Nous plaçons de faibles attentes dans cette lecture intérimaire puisque GLPG2737 ne devrait pas atteindre des niveaux d'exposition maximaux en 2 semaines. Ceci, associé à la revue du partenariat initiée par AbbVie nous a incité à ne pas prendre en compte le programme dans notre valorisation.

Nous valorisons GLPG à EUR120/tire (potentiel de hausse de 25%). Le newsflow spécifique au filgotinib au cours des 6-12 prochains mois pourrait nous permettre de réévaluer notre valorisation à EUR135/titre. Avant la fin de l'année, l'annonce d'un accord relatif au programme portant sur la mucoviscidose ainsi que de plus amples détails sur le pipeline *early-stage* de la société lors du R&D day du 25 octobre devraient soutenir la création de valeur.

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Contents

Part 1: A de-risked play: Buy reiterated

FV EUR120 per share

In our view, the current share price is fully supported by filgotinib. Taking into account the rest of the pipeline which seems de-risked to us in the light of clinical data generated to date, we believe Galapagos should trade at EUR120 per share, pointing to 25% upside relative to the current share price.

Our base case of EUR120 per share includes no sales from cystic fibrosis as the partnership with AbbVie is under review as present. We have low expectations from the upcoming interim FALCON results, which are due to be released in coming weeks (early Q4 2018), thereby limiting short-term downside risk on the share price.

Results from the FINCH-1 and FINCH-3 trials for filgotinib in RA are expected in H1 2019 and should be followed by results from the MANTA male testicular toxicity trial, enabling Gilead to file for approval with the FDA in RA towards H2 2019. Alongside positive results from pivotal trials in the UC and CD indications, we see a short-term bull case for filgotinib increasing our FV per share to EUR135, representing c.40% upside to the current level. Note that this case is based on filgotinib developments only and does not take into account the company's early stage clinical assets with more than 20 programmes in inflammatory diseases and fibrosis, including some already known targets that are set to enter clinical trials over the next 6-18 months. Additional late stage assets in IPF, AD and OA should drive significant value creation beyond 2019.

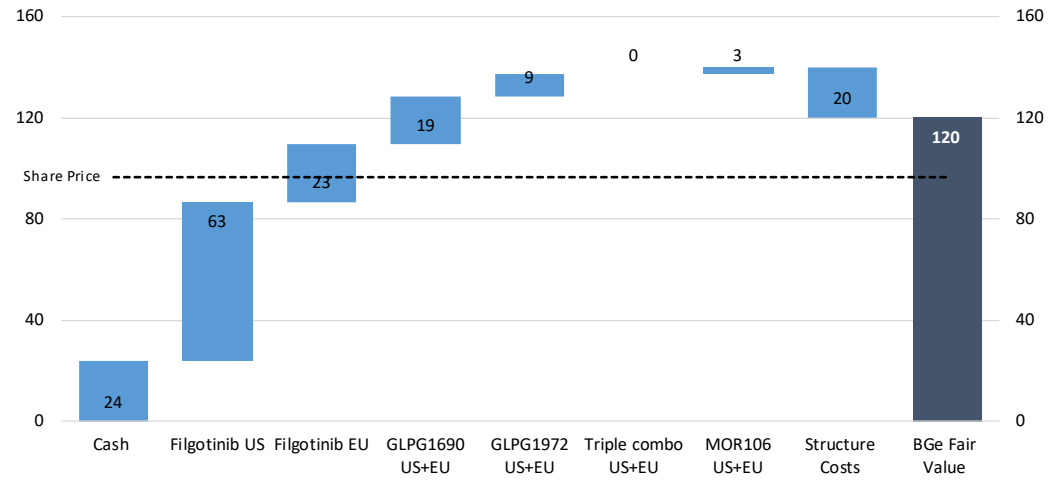
Fig. 1: Galapagos valuation (BGe, EURm)

Product	Partner	Region	Indication (incl.)	Non adj. PS	PoS	Risk adj. PS	Royalties	EV	/share
Filgotinib	Gilead	US	8 included in model	5 100	20%-80%	3 300	20%-30%	3 391	63
Filgotinib	Gilead	EU	8 included in model	1 600	20%-80%	900	20%-30%	1 234	23
GLPG1690	-	US+EU	IPF	2 100	60%	1 260	-	1 208	22
GLPG1972	Servier (EU)	US+EU	OA	3 000	30%	900	8% EU - 20% US	245	5
Triple combo	AbbVie	US+EU	CF	0	0%	0	15%-20%	0	0
MOR106	MorphoSys	US+EU	AD	1 100	40%	440	50/50 of 12%-22%	145	3
Cash								1 285	24
Structure								-1 042	-19
BGe FV								**6 465**	**120**

Peak sales rounded to the nearest tenth of a million.

Sources: Bryan Garnier & Co ests.

Fig. 2: Valuation waterfall chart



May not foot due to rounding differences.

Sources: Bryan Garnier & Co ests.

Upcoming newsflow

Fig. 3: Selected catalysts

Timing	Product	Partner	Indication	Phase	Trial name/details
Q4 2018	GLPG2451+GLPG2222+GLPG2737	AbbVie	CF	2	FALCON interim results
Q4 2018/Q1 2019	MOR106	MorphoSys/Novartis	AD	2	Trial expansion following bridging phase I trial
Late 2018/early 2019	-	AbbVie	CF	-	Review of the partnership with AbbVie
H1 2019	Filgotinib	Gilead	RA	3	FINCH-3 results
H1 2019	Filgotinib	Gilead	RA	3	FINCH-1 results
2019	Filgotinib	Gilead	UC	2	MANTA testicular toxicity initial readout
2019	MOR106	MorphoSys/Novartis	AD	2	-
2019	GLPG1205	-	IPF	2	PINTA results
H2 2019	Filgotinib	Gilead	RA	-	Regulatory filing
late 2019/early 2020	Filgotinib	Gilead	UC	3	SELECTION-1 results
late 2019/early 2020	Filgotinib	Gilead	CD	3	DIVERSITY-1 results
2020	Filgotinib	Gilead	UC	-	Regulatory filing
2020	Filgotinib	Gilead	CD	-	Regulatory filing
2020	Filgotinib	Gilead	SBS	2	-
2020	Filgotinib	Gilead	Fistulizing CD	2	-
2020	GLPG1972	Servier (EU)	Knee OA	2	ROCCELLA results
2021	GLPG1690	-	IPF	3	ISABELA-1 and ISABELA-2 results

Sources: Bryan Garnier & Co ests, Galapagos.

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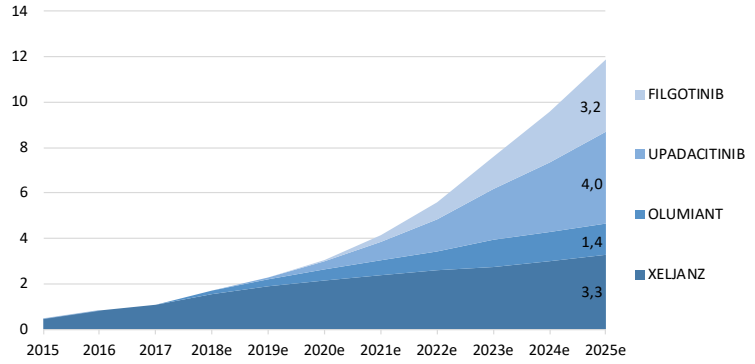
Part 2: Filgotinib blockbuster status de-risked

We see multi-billion blockbuster potential of >EUR6bn for best-in-class JAK-inhibitor filgotinib. While RA is set to be the main indication for the drug, accounting for c.50% of our peak sales towards the end of the next decade, seven additional indications currently being studied in either phase III or phase II trials should contribute to our remaining EUR2.5bn sales estimate. By 2025e already, we estimate filgotinib should have caught up with most of the competition in the JAK space.

Fig. 4: JAK-inhibitors sales (in EURbn, all indications)



Sources: Bryan Garnier & Co ests., Bloomberg (as of 01/10/18).

RA is a flagship indication with peak sales of EUR2.5bn

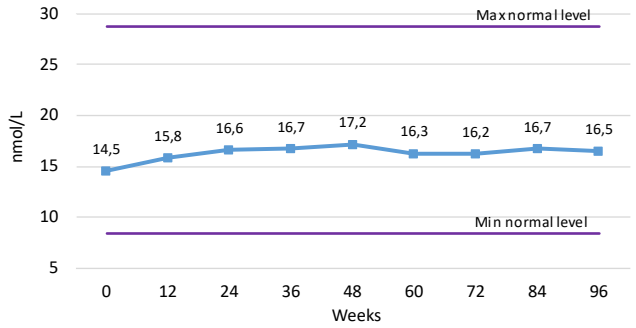
While the readout of the FINCH 2 phase III programme evaluating filgotinib on top of methotrexate has de-risked filgotinib in our view, bear in mind that three other trials need to be completed before Gilead could file for approval with the FDA. The FINCH 1 and FINCH 3 trials evaluating filgotinib in MTX-IR and as a monotherapy in MTX naive patients respectively should readout in H1 2019.

The third trial, a phase II study carried out to assess the testicular safety profile of filgotinib in UC patients (MANTA trial NCT03201445) is enrolling patients and is expected to read out in H1 2019 (primary endpoint at 13w). By the time the MANTA trial reads out (initial readout), the FINCH 1 and FINCH 3 should have also reported top-line results, enabling Gilead to file for approval in the US and in Europe.

We see the MANTA trial as a key catalyst for filgotinib. Despite not investigating filgotinib in RA, it should be the last trial enabling Gilead to finalise its data package for regulatory filing in the US. Indeed, a decrease in sperm concentration was observed in pre-clinical trials carried out in dogs and prompted Gilead to further investigate this particular issue. The trial is currently recruiting participants (n target = 250). In September, Gilead made a push by adding eight sites, including four in India and three in Ukraine, bringing the total number to 63 (vs. 28 in January 2018). It is our understanding that additional centres will be opened to further accelerate recruitment for this study.

We remain confident in the best-in-class safety profile of filgotinib and do not believe that the label will mention testicular toxicity. Indeed, 1/ pre-clinical studies showed that the decrease in sperm count was reversible, 2/ stable testosterone levels in the 14.5 to 17.2nmol/L range were measured in males recruited in the DARWIN 1, 2 and 3 trials (normal levels for males >18yo 8.4-28.7nmol/L). John McHutchison, Gilead's CSO stated during the Q2 call that he believes the "margin is adequate above and beyond minor histological abnormalities seen in preclinical models". Lastly, we would point out that RA more commonly affects women (3:1 ratio) between the ages of 40 and 60.

Fig. 5: Total testosterone (measured in males recruited in DARWIN1, 2 and 3)



Sources: Galapagos.

Lastly, we would not read into the pharmacokinetics trial being conducted in patients with impaired hepatic function, since filgotinib has already demonstrated that its use does not trigger an elevation in AST and ALT levels.

We believe that penetration of the JAK inhibitor class has been affected by the poor safety profile of assets having been approved so far, pointing to the occurrence of AEs such as thrombotic events, opportunistic infections and herpes zoster risks primarily. The best-in-class profile of filgotinib, for which we model peak sales of EUR3bn in RA, should help expand the class in our view. The results from the FINCH 2 trials confirmed our view.

- Conducted in 423 RA patients non-responders to biologics and randomised on a 1:1:1 basis to either filgotinib 100mgQD, 200mgQD or placebo, the efficacy results were strong with the 100mgQD dose achieving ACR20, ACR50 and ACR70 responder rates (adj. for placebo) of 26.4%, 17.1% and 7.6% respectively at 12 weeks. The dose response profile was also clear with the 200mgQD dose achieving responder rates of 34.9%, 28% and 15% within the same timeframe. At week 24, ACR20, ACR50 and ACR70 responder rates (adj. for placebo) stood at 20.4%, 16.4% and 12.2% for the 100mgQD dose, and at 34.9%, 26.7% and 23.9% for the 200mgQD dose. The efficacy plateau at 24 weeks on ACR20 and ACR50 responder rates while the ACR70 response rate continued to progress across all doses suggested that ACR50 responders at 12 weeks improved their response by week 24, which bodes well for longer term data, bearing in mind that the FINCH-1 and FINCH-3 trials are evaluating filgotinib at 52 weeks. All secondary endpoints including low disease activity and clinical remission were also

met, standing at 13.9% and 18.4% at 24 weeks for the 100mgQD and 200mgQD doses respectively.
Comparing filgotinib to other JAKs, we noted that the efficacy profile is a touch lower than ABBV's upadacitinib albeit higher than that of PFE Xeljanz (tofacitinib) and LLY Olumiant (baricitinib). The same conclusion could be drawn from low disease activity (LDA) and remission responder rates.

Fig. 6: ACR responder rates (12w, active delta vs placebo in %)

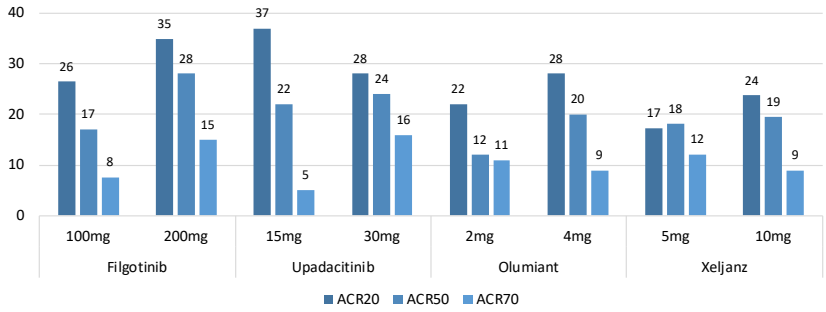
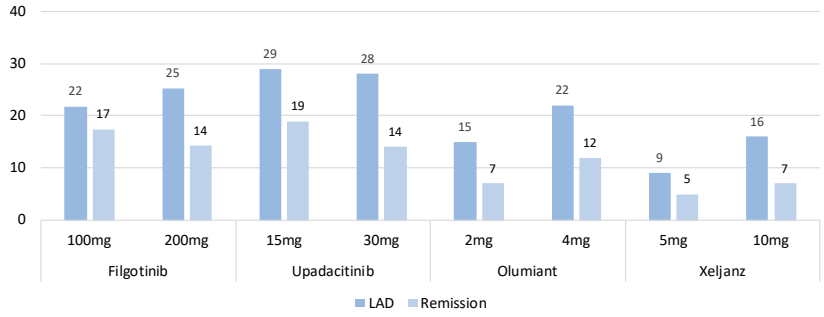


Fig. 7: Low disease activity (LDA) and remission rates (12w, active delta vs placebo in %)



Not H2H trials

Sources: Galapagos (FINCH 2), AbbVie (SELECT-BEYOND), Eli Lilly (RA-BEACON), Pfizer (NCT00960440).

- Turning to safety which is key for JAK inhibitors in the market or in development, the safety profile of filgotinib is clean. Indeed, SAEs occurred in 3.4% 5.2% and 4.1% of patients in the placebo, 100mgQD and 200mgQD dose groups respectively with no imbalances for patients discontinuing the trial in between the three groups. The incidence of uncomplicated herpes zoster was low in the filgotinib groups (1.3% and 1.4%), lower than that seen with baricitinib and baricitinib (Kevin L. Winthrop et al., 2016 and 2017 publications) and in line with that of upadacitinib in our view. Only one MACE occurred in the filgotinib 100mgQG group (myocardial ischemia). While DVT and PE, alongside opportunistic infection events are a major concern for the JAK class, none occurred in both filgotinib groups. This is important and should reassure, especially since one DVT occurred in the TORTUGA phase II study released in early September and evaluating the efficacy of filgotinib in AS. We look forward to confirmation of the safety profile in the two additional trials from the phase III

programme carried out in larger patient populations (1,650 and 1,200 patients for FINCH-1 and 2 respectively).

Depending on whether a priority voucher is be used in this indication, we estimate approval could occur in early 2020 or H2 2020 respectively. We have modelled first sales of EUR33m in 2020 in the US (first EU sales one year later in 2021), almost equally split in between 1L and 2-3L and consider this figure conservative in light of 1/ LLY Olumiant's USD44m in sales achieved in one month only following approval on 1st June 2018 (LLY US Q2 results) and 2/ ABBV upadacitinib USD81m sales expectations for 2019 upon approval in late H2 2019.

UC and CD to fuel growth

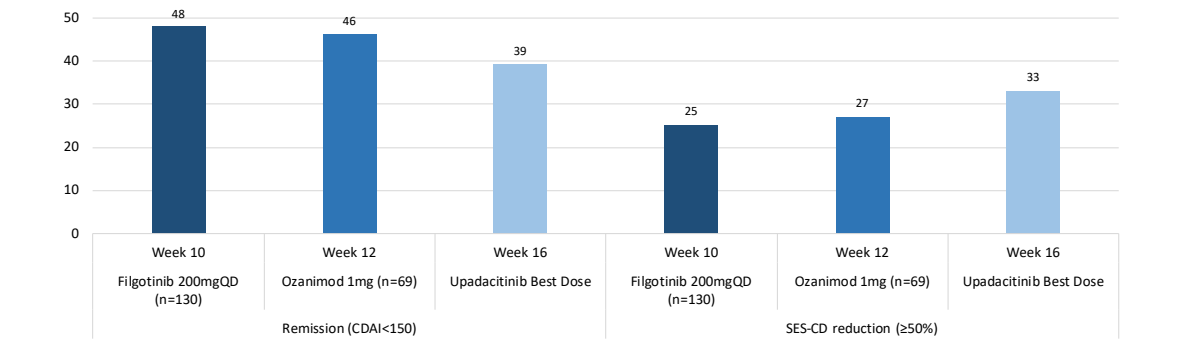
We view UC and CD as two key indications for filgotinib alongside RA as we estimate combined sales from these two indications should represent 20% of our peak sales for the product candidate, or EUR1.2bn. The DIVERSITY1 and SELECTION1 trials in CD and UC respectively should both read out in late 2019/early 2020.

First mover advantage in CD may be the best only solution

In Crohn's disease, Gilead/Galapagos could have the first mover advantage with an oral route should Gilead decide to use one of its vouchers. There is a clear window of opportunity for both partners as upadacitinib phase III trial from ABBV should read out in late 2019 and especially since Celgene, following the discontinuation of mongersen (GED-0301), is now left with ozanimod (apremilast, S1P inhibitor), which is not set to report phase III results in CD before H1 2020.

Despite the difference in patient populations included in the trials carried out over different timeframes (e.g. 58%, 54% and 95% of TNF failure patients in the FITZROY, STEPSTONE and CELEST trials respectively) filgotinib appears competitive in CD in our view.

Fig. 8: Remission and SES-CD≥50% responder rates (%)

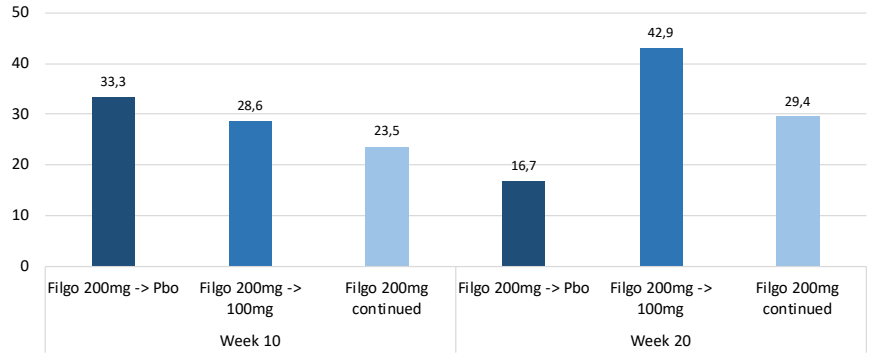


Not H2H trials

Sources: Galapagos (FITZROY), Celgene (STEPSTONE), AbbVie (CELEST).

48% of patient were in clinical remission (CDAI<150, p <0.05) which is higher than what ozanimod and upadacitinib achieved at 12 and 16 weeks respectively. For endoscopic remission (SES-CD≤50%), we believe that the efficacy gap vs ozanimod and upadacitinib could be narrowed overtime. Indeed, filgotinib's clinical response was maintained through to week 20 in the FTZROY trial with 50% to 71% of initial responders at 200mg showing clinical remission, despite steroid tapering from week 10 onwards (poster presented at DDW 2017). Against this backdrop, the significant decrease of ≥50% in inflammatory markers (CRP and calprotectin) reported in 27% of patients in the active arm at week 10 and their continuous normalisation (see chart below) through to week 20 is encouraging and may well be a first step towards an increased endoscopic response over a longer period.

Fig. 9: CRP normalisation (FITZROY 20w, ITT-LOCF)



Sources: Galapagos (in subjects with high baseline CRP>8mg/L).

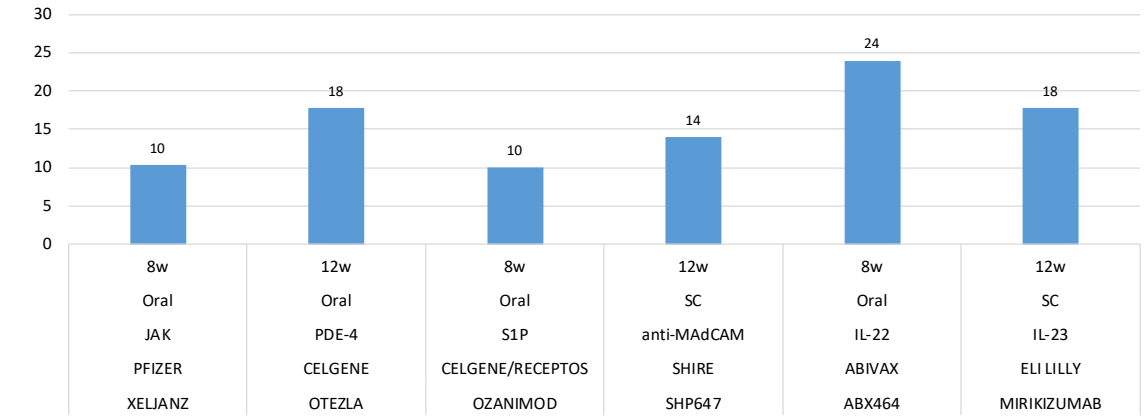
As mentioned above, safety is likely to be the key for filgotinib. No imbalance was found between the placebo and the active groups at week 10. As in RA, filgotinib had no impact on LDL levels while a positive impact on HDL levels was noted. Also to note was that compared with upadacitinib, which showed an increase in Alanine Aminotransferase levels, a marker of liver damage, no changes were seen with filgotinib. If the safety profile is confirmed in the 58-week DIVERSITY 1 trial, it would be a best-in-class status in our view. Indeed, upadacitinib might suffer from a high rate of infections, ranging from 26.1% to 37.3% depending on the dose, compared with 3% for filgotinib at 20 weeks as well as concerns over GI perforation (one event) and MACE events (two events), which occurred in phase II.

UC becoming increasingly competitive

While JAK inhibitors are one step ahead of other development programmes, the UC field is becoming increasingly competitive with new treatment alternatives putting the bar high. The last entrant was Abivax with strong results from a phase IIa trial of ABX464 (anti IL-22) released at the beginning of September 2018 and showing 1/ a clinical remission rate of 35% (24%

adjusted for placebo) and 2/ 50% of patient with colorectal mucosal healing as early as eight weeks (39% adjusted for placebo).

Fig. 10: Remission rate (placebo adjusted, %)



Sources: Pfizer, Celgene, Celgene/Receptos, Shire, Abivax, Eli Lilly.

No comprehensive data has been disclosed by Galapagos/Gilead who are currently conducting a phase IIb/III trial. In May 2018, the SELECTION study progressed in phase III following a positive DSMB recommendation based on a futility analysis carried out in 350 patients who completed the phase IIb induction part of the trial, triggering a USD15m milestone payment for GLPG.

New oral alternatives like Tyk2 inhibitors are emerging in the field of autoimmune diseases especially UC. Based on the available data, it appears that this class has the potential to answer safety issues faced by some JAK inhibitors as they do not hit JAK1 and JAK3. If some products from this class reach the market (not before 2021/2022), we do not expect them to have a negative effect on the entire JAK class. Indeed, JAK inhibitors Xeljanz and filgotinib will be on the market for a couple years, each with its specific marketing message. Against this backdrop, our thought is that filgotinib would have had enough time to gain recognition for its best-in-class safety profile and should not be affected, unlike baricitinib, Xeljanz and upadacitinib showing a poor safety profile. Although the safety profile of Tyk2 might be better, it seems to us that a trade-off with efficacy would be needed, explaining why some assets have been discontinued, notably by Pfizer.

In all, we remain relatively cautious in terms of market share for JAK inhibitors on the UC market (BGe 15%), although filgotinib should be the more widely used drug in this class with a market share of 30%.

Recently de-risked, 'satellite' indications are key

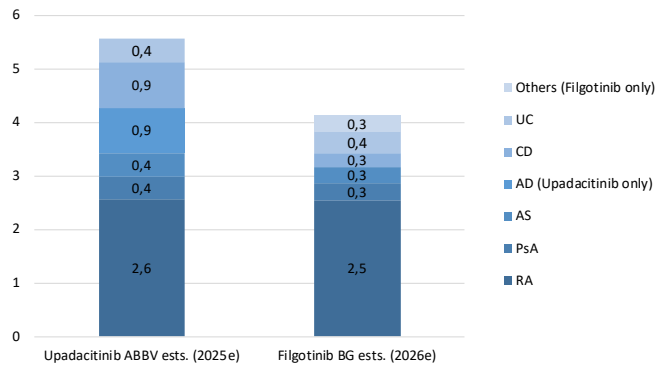
What made Humira a blockbuster was not only the marketing firepower AbbVie put behind the drug, but also the nine indications for which it is approved alongside RA, making it a reference product for rheumatologists who often face patients with multiple concurrent diseases.

Galapagos/Gilead are clearly following this strategy with a recent win in Ankylosing Spondylitis. At 12 weeks, the TORTUGA phase II study, evaluating filgotinib in 126 patients across European centres, reached its primary endpoint with filgotinib 200mg achieving a greater improvement in the AS disease activity score vs placebo (-1.5 vs -0.6, p<0.0001). The secondary endpoint showed that 76% of filgotinib patients achieved an ASAS20 score vs 40% for the placebo. Although these results need to be confirmed in a phase III trial and over a longer period of time, note that the placebo-adjusted ASAS20 responder rate of 36% appears very competitive to the c.30% placebo-adjusted response rate shown by secukinumab in the MEASURE 1 trial over a slightly longer period (i.e. 16 weeks). Turning to safety, the occurrence of AEs was similar in both arms. It is worth mentioning that one patient in the filgotinib group had pneumonia which resolved following antibiotic treatment. However, we would not read into the non-serious deep venous thrombosis case experienced by one patient in the filgotinib group as the latter had an inherited risk of thrombosis. We consider these results as very good news for Galapagos and Gilead, increasing confidence in the potential of filgotinib. While the TORTUGA phase II trial was carried out in Europe only, including US sites in the phase III trial due to start in early 2019 should not be an issue in our view.

Note also that the results from the EQUATOR phase II trial in Psoriatic Arthritis (PsA), released at the beginning of the year also support the long-term growth story for filgotinib. At week 16, ACR20, ACR50 and ACR70 scores stood at 80% (p<0.001), 48% (p<0.001) and 23% (p<0.01) vs 33%, 15% and 6% for the placebo, respectively.

With eight indications included in our model on top of which three represent free upside to our estimates (cutaneous lupus, lupus nephropathy and uveitis), we are confident in filgotinib's ability to reach EUR4.7bn in sales six years after launch (i.e. 2026) especially since AbbVie expects to derive sales of EUR5.5bn from upadacitinib about six years after launch i.e. in 2025.

Fig. 11: ABBV upadacitinib and GLPG/GILD filgotinib sales estimates 6y post-launch (EURbn)



Sources: Bryan, Garnier & Co ests, AbbVie's internal estimates (2017).

Estimates below are non-risk adjusted sales for filgotinib by indication included in our model.

Fig. 12: Filgotinib sales in inflammatory diseases (BGe, EURm)

	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
TOTAL	54	295	751	1 418	2 234	3 180	4 123	4 720	5 318	5 673	5 950
% growth		445%	155%	89%	58%	42%	30%	14%	13%	7%	5%
RA	53	273	590	989	1 469	2 033	2 542	2 751	2 956	3 042	3 130
% growth		417%	117%	68%	49%	38%	25%	8%	7%	3%	3%
% total	97%	92%	79%	70%	66%	64%	62%	58%	56%	54%	53%
UC	1	13	46	101	177	276	399	491	589	661	735
% growth		1473%	252%	117%	76%	56%	45%	23%	20%	12%	11%
% total	2%	4%	6%	7%	8%	9%	10%	10%	11%	12%	12%
CD	1	9	32	69	121	188	271	333	398	448	498
% growth		1455%	246%	115%	75%	56%	45%	23%	20%	12%	11%
% total	1%	3%	4%	5%	5%	6%	7%	7%	7%	8%	8%
Fistulizing	0	0	0	17	41	67	94	121	151	178	190
% growth					148%	62%	40%	30%	24%	18%	7%
% total				1%	2%	2%	2%	3%	3%	3%	3%
SBS	0	0	0	6	16	26	36	47	59	70	75
% growth					150%	63%	41%	30%	24%	19%	7%
% total				0%	1%	1%	1%	1%	1%	1%	1%
AS	0	0	39	97	158	223	290	361	428	460	474
% growth				148%	63%	41%	30%	24%	18%	7%	3%
% total			5%	7%	7%	7%	7%	8%	8%	8%	8%
PsA	0	0	43	108	176	248	323	402	477	511	528
% growth				150%	63%	41%	30%	24%	19%	7%	3%
% total			6%	8%	8%	8%	8%	9%	9%	9%	9%
SjS	0	0	0	31	76	121	167	214	261	304	320
% growth					144%	60%	38%	28%	22%	16%	5%
% total				2%	3%	4%	4%	5%	5%	5%	5%

Sources: Bryan, Garnier & Co ests.

Why buying a drug when the whole company costs about the same?

We understand that Gilead's board would like the company to expand more aggressively in areas other than antivirals, potentially explaining the departure of the CEO who is due to step down at the end of 2018. For Gilead, top management changes would be an opportunity to re-evaluate its M&A options with USD13.2bn in cash and cash equivalents as of June 2018, especially regarding Galapagos since 1/ Gilead has a 12.5% stake in the company and 2/ the standstill agreement between the two companies expired on 31st December 2017.

Galapagos' exercise of its option to co-promote filgotinib in eight EU countries in late 2017 means it is now more likely that Gilead could decide to explore strategic options regarding filgotinib to avoid 1/ a high royalty rate on US sales in the 20-30% range and 2/ a profit split in the eight European countries while assuming 65% of co-promotion spending.

A spin-off would be (too) expensive. The latter transaction would be fully justified if Gilead believes that it could drive filgotinib sales higher on a standalone basis (e.g. rationale behind the JnJ/Actelion deal). However, it is not likely to be the case in our view as Gilead is already responsible for US commercial efforts. As a result, we consider the premium to be paid as relatively low (BGe ≤20%). Indeed, a portion of the extra sales that could be generated would not need to be paid to GLPG shareholders. This transaction would value filgotinib in the region of EUR4.9-5.3bn or >93% of Galapagos current market cap (see table below).

Fig. 13: Spin-off of filgotinib to GILEAD: transaction value table (net of GILD participation)

EURbn		2018	2019	2020	2021	2022
	0%	4,1	4,5	4,8	5,1	5,4
	5%	4,3	4,7	5,0	5,4	5,7
Premium	10%	4,5	4,9	5,3	5,6	6,0
	15%	4,7	5,1	5,5	5,9	6,2
	20%	4,9	5,3	5,7	6,1	6,5

/share		2018	2019	2020	2021	2022
	0%	76	83	89	95	100
	5%	80	87	93	99	105
Premium	10%	84	91	97	104	110
	15%	88	95	102	109	115
	20%	92	99	106	114	120

Sources: Bryan, Garnier & Co ests.

From a financial and strategic perspective it would make more sense for Gilead to acquire Galapagos in our view: 1/ from a financial standpoint, current levels of c.EUR100/share do not take into account the company's late-stage pipeline or any M&A speculative premium, 2/ from a strategic standpoint, Galapagos would enable Gilead to diversify beyond filgotinib in rheumatic diseases with GLPG1972 in OA and in respiratory diseases with GLPG1690 in IPF.

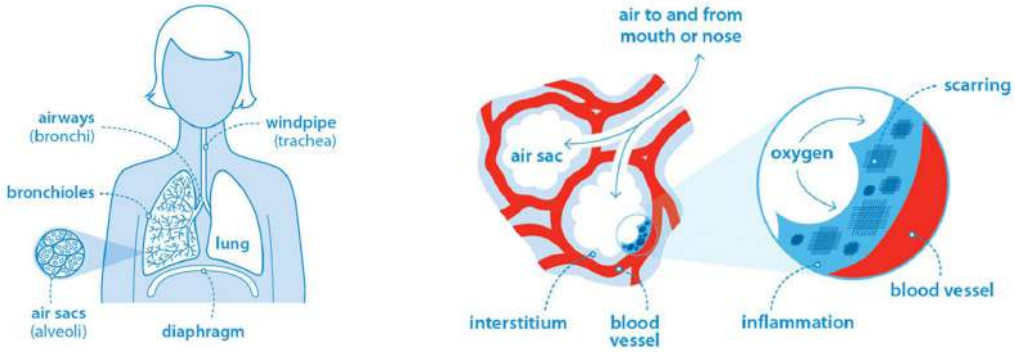
Part 3: Proprietary GLPG1690 is big but overlooked

Worse than many cancer types

Idiopathic Pulmonary Fibrosis (IPF) is a type of irreversible and fatal lung disease resulting in fibrosis (scarring) of the lungs that gets worse over time. While many lung diseases affect the airways or blood vessels, IPF involves the interstitium, a network of fluid-filled space composed of water and solutes found in tissue layers lining the lungs (gut, muscles…) acting as a membrane between the air sacs and the blood vessels. Note that the interstitium also acts as a reservoir and transportation system for nutrients and solutes distribution between organs, signalling molecules and cells participating in immune regulation.

As the interstitium scars overtime, it limits the amount of oxygen that can get into the blood, making it harder for patients to breathe and obtain enough oxygen to carry out day-to-day activities. Main symptoms include shortness of breath, coughing and fatigue. Some common risk factors for IPF have been identified with 75% of patients suffering from the disease being current or previous smokers and/or having gastroesophageal reflux disease (GERD). However, its cause is not yet fully understood, hence the name "idiopathic".

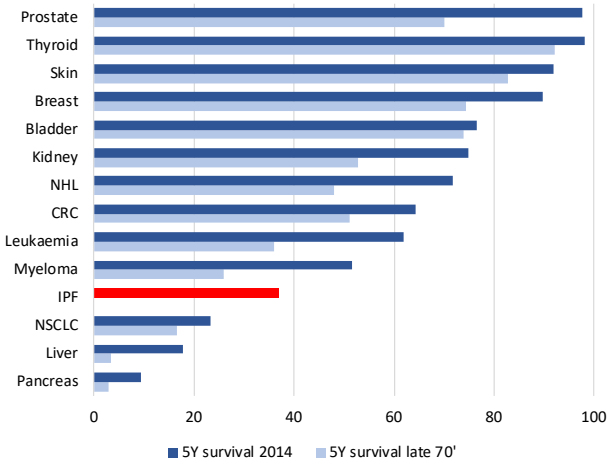
Fig. 14: Idiopathic Pulmonary Fibrosis



Sources: British Lung Foundation.

IPF is an orphan disease with an estimated prevalence of 150,000 patients suffering from the disease in the US and in Europe (Top 5 countries). It primarily affects men (approx. 75%) over the age of 50, although around 85% of diagnoses are made for people over 70. It has a poor prognosis. Indeed, the 5-year median survival rate of around 30% compares to that of the deadliest cancer types.

Fig. 15: 5-year survival rate evolution for some cancer types (%)



Sources: Bryan, Garnier & Co ests; NIH.

Current SoC faces several hurdles

The FDA approved Roche's Esbriet (pirfenidone) after a first CRL in May 2010 and Boehringer Ingelheim's OFEV (nintedanib) in 2014. Despite the high unmet medical need, both drugs had a slower than expected uptake due to their 1/ limited efficacy and 2/ poor safety profile.

Both drugs slow progression of the disease but do not halt it. Despite having reached their primary endpoint of mean forced vital capacity decline (FVC) vs placebo in phase III, FVC at 52 weeks was significantly lower than that at baseline and stood at -235mL for Esbriet at the 2403mgQD dose in the ASCEND trial (vs -428mL for placebo) and -115mL for OFEV at the 150mg/BID dose in the INPULSIS-1 trial (vs -240mL for placebo).

However, this slowdown in progression of the disease was only achieved at the expense of increased safety risks with 15% and 21% of adverse events leading to discontinuation for Esbriet and OFEV vs 10% and 15% for placebo respectively. Note that the labels for both drugs point to 1/ increased risk of gastrointestinal side effects including nausea (Esbriet 36% vs 16% pbo, OFEV 24% vs 7% pbo), abdominal pain (Esbriet 24% vs 15% pbo, OFEV 15% vs 6% pbo), diarrhoea (Esbriet 26% vs 7% pbo, OFEV 62% vs 18% pbo), vomiting (Esbriet 13% vs 6% pbo, OFEV 12% vs 3% pbo) and dyspepsia for Esbriet (19% vs 7% pbo). The latter measure does not appear on OFEV's label, with the drug's poor safety profile nevertheless exacerbated by two gastrointestinal perforations. On top of that, OFEV's safety profile is further burdened by liver toxicity (14% vs 3% pbo) while Esbriet's highlighted photosensitivity (9% vs 1%).

Despite efficacy and safety limitations, significant marketing efforts including co-pay programmes are nevertheless bearing fruit with sales now expected to reach EUR830m and EUR981m in 2018 for Esbriet and OFEV respectively (*source: Bloomberg*). However, in a real-life setting, many patients are reluctant to start their treatment and even when they do, approx. 25% of them discontinue it each year. In all, it is estimated that less than 55% (80,000) of the 150,000 diagnosed patients are on IPF-approved therapies.

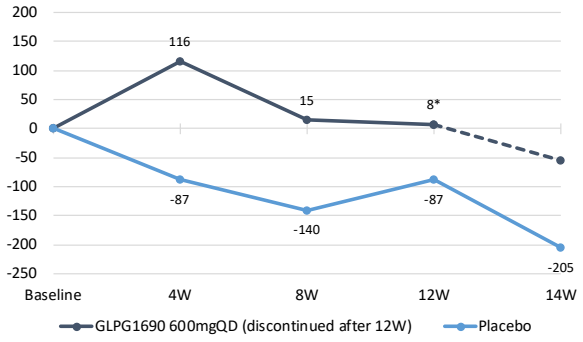
GLPG1690 could reshuffle the cards in IPF

In August 2017, Galapagos reported very encouraging results from the FLORA phase IIa trial evaluating the safety of oral autotaxin inhibitor GLPG1690. Autotaxin (ATX) is a secreted enzyme involved in the production of lysophosphatidic acid (LPA) whose signals can activate the production of pro-fibrotic genes (e.g. IL-6 and connective tissue growth factor, CTGF) causing the interstitium to scar over time. The inhibitory effect of GLPG1690 on IPF lung fibroblasts has not only been demonstrated in a preclinical model but it has also been confirmed in recent clinical trials.

The FLORA phase IIa trial randomised 23 IPF patients to follow a 12-week treatment course of GLPG1690 at the 600mgQD dose (n=17) or placebo (n=6). Although not powered to show efficacy, GLPG showed a statistically significant +8mL increase in FVC change from baseline (CFB) compared with the placebo at 12 weeks (p <0.05). These results are promising, especially since the -87mL FVC CFB in the placebo group compares well with data from a larger phase III trial carried out by Roche (ASCEND, placebo -95mL) and Boehringer Ingelheim (INPULSIS-1, placebo -70Ml CFB). Note that using the LOCF analysis method, comparable results would have been a 25mL increase vs 70mL for placebo. GLPG1690's potential to halt disease FVC decline was further supported by functional respiratory imaging (FRI) data, presented at ATS 2018. Despite not increasing lobar volume (p=0.5153), which would advocate a reversal of the disease's course, a statistically significant change in specific airway volume (p=0.0181) and specific airway resistance (p=0.0334) at total lung capacity was shown.

Acknowledging that the sample size was small (n=23 and three patients in the placebo group evaluable for FRI), the FLORA trial suggests that GLPG1690 is ≥1.5x more potent than Esbriet or OFEV in our view.

Fig. 16: FLORA Phase IIa efficacy results (FVC change vs baseline, mL)



** SS p <0.05*

Sources: Galapagos.

Turning to safety, GLPG1690 stands out as well. While AEs were balanced in-between the active and placebo arm, SAEs were significantly lower for GLPG1690 (6% vs 33%) and only 6% of patients in the active discontinued compared with 17% in the placebo group. The rate of gastrointestinal side effects was particularly low at 6% vs 33% for the placebo group.

Fig. 17: Clinical trial results in IPF: Esbriet, OFEV, GLPG1690

	ROCHE		Boehringer Ingelheim		GALAPAGOS	
	ESBRIET pirfenidone pyridone ASCEND Ph III		OFEV nintedanib TKI INPULSIS-1 Ph III		GLPG1690 na autotaxin inhibitor FLORA Ph IIa	
dose	2403mgQD	pbo	150mgBID	pbo	600mgQD	pbo
n	278	277	309	204	17	6
FVC (MEAN CFB IN mL)						
4W			3	-8	116	-87
pbo adj.			10		203	
p-value					NS	
8W					15	-140
pbo adj.					155	
p-value					<0,05	
12W	-31	-95	-18	-70	8	-87
pbo adj.	64		53		95	
p-value					NS	
14W (GLPG1690 disc. 12W)					-55	-205
pbo adj.					150	
p-value					NS	
52W	-235	-428	-115	-240		
pbo adj.	193		125			
p-value						
SAFETY (%)						
AE	97%	91%	94%	87%	65%	67%
SAE	20%	25%	31%	27%	6%	33%
AE discontinuation	15%	10%	21%	15%	6%	17%
AE death	4%	7%			0%	0%
GI - Nausea	36%	16%	24%	7%		
GI - Abdominal pain	24%	15%	15%	6%		
GI - Diarrhea	26%	7%	62%	18%	6%	33%
GI - Vomiting	13%	6%	12%	3%		
GI - Dyspepsia	19%	7%			12%	17%
Liver (incl. ALT)	4%	1%	14%	3%		

Efficacy: INPULSIS-1 4W, 12W and ASCEND 12W data estimated from chart.

Safety: Pooled data from the TOMORROW, INPULSIS-1 & 2 trials for Esbriet. Pooled data from the ASCEND, CAPACITY-1 & 2 for OFEV.

Sources: Roche, Boehringer Ingelheim, Galapagos.

On the back of this strong set of results, Galapagos announced in Q3 the initiation of the ISABELA phase III programme, which consists of two trials, ISABELA-1 and -2 that will start enrolling by the end of the year (not on clinicaltrial.gov yet). A total of c.1,500 patients will be randomised to GLPG1690 dose 1, GLPG1690 dose 2 or placebo on top of SoC (i.e. Esbriet or OFEV). While the primary endpoint is change in FVC (mL) at 52 weeks, all patients will be treated until the last one passes the final visit. The length of the trial should enable Galapagos to build a consistent database notably on side effects but also to go after secondary endpoints such as hospitalisation and mortality potentially. The ISABELA phase III programme is not likely to read out before early 2021 as we expect recruitment to last 12-18 months. However, strong support from 1/ the

medical community, highlighted by the FDA authorising over >1,500 patients in the phase III programme on the back of a 23-patient phase IIa and 2/ the high engagement of patient advocacy groups that are looking for alternative treatments could lead to a shorter recruitment period, hence a potential read-out in late 2020 although we are not ruling out the possibility of Galapagos adding an interim look into the design of the trial.

We do not see Fibrogen's IV pamrevlumab as a threat. Pamrevlumab is an anti-CTGF antibody that reported phase II results in August 2017. 103 patients were randomised (1:1 basis) to either IV pamrevlumab 30mg/kgQ3W on top of SoC (n=57) or to placebo. At 48 weeks (16 infusions), the results were encouraging with patients in the active arm group having an average decrease in FVC of 129mL (-2.85% CFB) compared with a 308mL (-7.17% CFB) decrease for patients in the placebo group (179mL pbo adjusted, p=0.038). Although this data is encouraging, pamrevlumab does not halt progression of the disease. While the FDA granted pamrevlumab fast track designation in IPF, Fibrogen has not communicated on its phase III plans yet.

>EUR2bn peak sales estimate

Following positive data from the ISABELA programme, we would expect GLPG1960 to be prescribed as an add-on to SoC and as a standalone treatment i.e. monotherapy with first sales in 2022 following a priority review for the product candidate, which already benefits from orphan status. Our model points to peak sales potential north of EUR2.1bn out of which 70% should be streamed from the US.

Fig. 18: GLPG1690 sales in IPF (EURm)

	2022	2023	2024	2025	2026	2027	2028	2029	2030
GLPG1690	32	396	769	1 150	1 541	1 940	2 078	2 100	2 123
% growth		1120%	94%	50%	34%	26%	7%	1%	1%
% ms (value)	1%	12%	22%	31%	40%	49%	54%	57%	60%
Europe	11	136	263	394	527	663	699	682	666
% growth		1096%	94%	49%	34%	26%	5%	-2%	-2%
% total	35%	34%	34%	34%	34%	34%	34%	32%	31%
US	21	260	506	757	1 014	1 278	1 379	1 418	1 458
% growth		1134%	94%	50%	34%	26%	8%	3%	3%
% total	65%	66%	66%	66%	66%	66%	66%	68%	69%

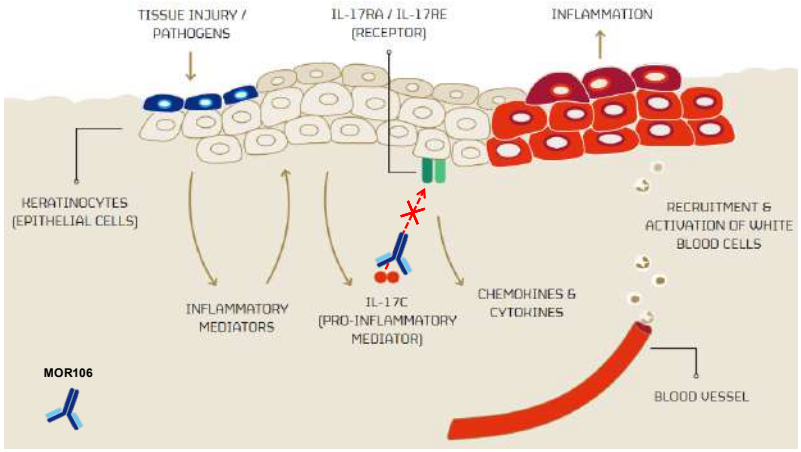
Sources: Bryan, Garnier & Co ests.

Part 4: the remaining pipeline consists of free options at the current level

MOR106 differentiated in a competitive indication

MOR106 is an antibody initially co-developed by MorphoSys and Galapagos, targeting Interleukin 17C (IL-17C). In July 2018, it was in-licensed by Novartis. Literature states that the IL-17C receptor is expressed much more on epithelial cells and so has a lower systemic exposure. Its mechanism of action allows the cytokine to have a direct effect on the IL-17A pathway but also a loop effect on Th17 cells, which made IL17C a local amplifier of inflammation in the skin. As such, by inhibiting IL-17C, MOR106 leads to a decrease in skin inflammation and potentially to an improvement in AD clearance.

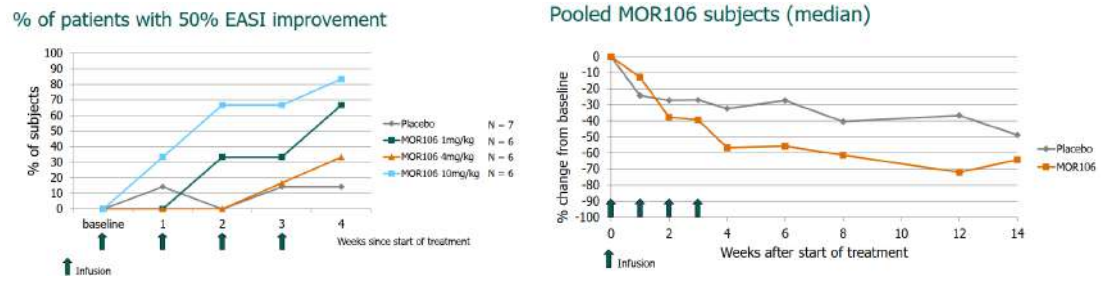
Fig. 19: MOR106 MoA (IL-17C pathway)



Sources: Adapted from MorphoSys.

The antibody was evaluated in a small phase I study (n=25) in patients suffering from atopic dermatitis randomised to IV MOR106 at the 1mg/kgQW, 4mg/kgQW and 10 mg/kgQW doses vs placebo. Top-line results, comparing nicely with dupilumab in our view, were communicated in Q1 2018. The available data showed a favourable safety profile and promising clinical efficacy. Indeed, 83% of patients (n=5/6) treated at the highest dose showed at least a 50% improvement in signs and symptoms of atopic dermatitis as measured by EASI-50 at week 4, compared with 17% (n=1/7) of patients in the placebo group. Important to note as well is that MOR106 maintained its effect almost three months (11 weeks) after discontinuation of treatment.

Fig. 20: MOR106: EASI-50 responder rate (left), EASI CFB (right)



Sources: Galapagos.

In May 2018, Galapagos and MorphoSys announced the initiation of the IGUANA phase II trial in patients with moderate-to-severe AD. 180 patients will be enrolled in this 12-week trial evaluating IV MOR106 at the 1mg/kg, 4mg/kg and 10 mg/kg doses administered every 2-weeks or 4-weeks vs placebo and which assess efficacy by the percentage change from baseline in Eczema Area and Severity Index (EASI). The IGUANA trial is expected to read out in late 2019.

While the collaboration signed in 2008 initially involved Galapagos managing the biology related to the targeted diseases and MorphoSys taking care of the engineering of the antibody, Novartis in-licensed MOR106 in July 2018 by agreeing to pay a EUR95m upfront, up to USD1bn in total milestones and if approved low-teens to low-20s royalty rates. MOR106 looks like a very nice complement to the dermatology franchise being built around Cosentyx, considering that Novartis also has an oral H4 receptor antagonist called ZPL389 acquired in December 2016 from Ziarco. We would expect the phase II trial for ZPL389 to start enrols its first patients shortly (NCT03517566).

More recently, in September 2018, Galapagos and MorphoSys announced the initiation of the bridging study for MOR106 which aims to assess the bioavailability of the antibody as a SC formulation (vs IV currently). This trial will be an important milestone for both companies, not only because it will be the last they will lead following an out-licensing deal to Novartis but also because we see the SC formulation as the last competitive gap in-between MOR106 and Sanofi's Dupixent.

Upon successful development and approval, we estimate MOR106 will hit the market in 2023 i.e. more than five years after the approval of Dupixent, which should have reached EUR5bn in sales by then (including EUR3.5bn in atopic dermatitis, BGe). As a result, and from the data available to us at present, we see the sweet spot of MOR106 in patients non responders to Dupixent. Our peak sales estimate stands at EUR1.1bn. Lastly, we are not ruling out the prospect of Novartis deciding to evaluate MOR106 in respiratory diseases.

Fig. 21: MOR106 Sales estimates (EURm)

	2023	2024	2025	2026	2027	2028	2029	2030
TOTAL SALES	**4**	**155**	**318**	**493**	**681**	**883**	**1 079**	**1 117**
% growth		**3396%**	**105%**	**55%**	**38%**	**30%**	**22%**	**4%**
Europe	3	55	112	174	239	309	364	364
% growth		2069%	103%	54%	38%	29%	18%	0%
% Sales	58%	36%	35%	35%	35%	35%	34%	33%
US	2	99	205	319	442	574	714	753
% growth		5204%	106%	56%	38%	30%	24%	5%
% Sales	42%	64%	65%	65%	65%	65%	66%	67%

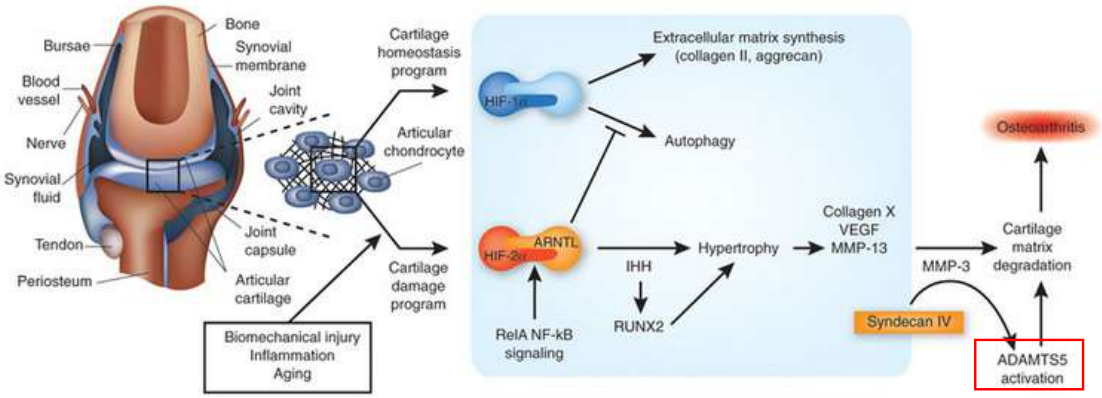
Sources: Bryan, Garnier & Co ests.

GLPG1972 in osteoarthritis could turn out to be a large opportunity

GLPG1972 was developed as part of the 2010 alliance signed with Servier, under which the latter gained the exclusive option to license any small molecules developed by Galapagos in the OA field after the completion of phase I trial. As part of the agreement, Galapagos was granted EUR7m in research funding and remains eligible to EUR290 in milestone payments as well as high-single digit royalties on net sales in Europe (BGe 8%). This deal is very attractive for Galapagos, which remains only responsible for running the US part of the trials paid for by Servier and retains full rights in the US.

The first molecule to reach the clinic as part of the alliance described above, GLPG1972 targets ADAMTS-5, an enzyme encoding for the ADAMTS5 gene and whose catalytic domain deletion (deletion of part of the protein chain which contains the region where the chemical reaction takes place) was linked with cartilage destruction resistance in osteoarthritis model (Sonya S. Glasson et al., 2005). Initially developed as an oral solution, a bioavailability trial enabled to bring GLPG1972 to phase I in the form of a tablet. On top of potentially being the first disease modifying osteoarthritis drug (DMOAD) the tablet form could provide a significant edge in the osteoarthritis space as pain management in patients is mostly carried out through injections.

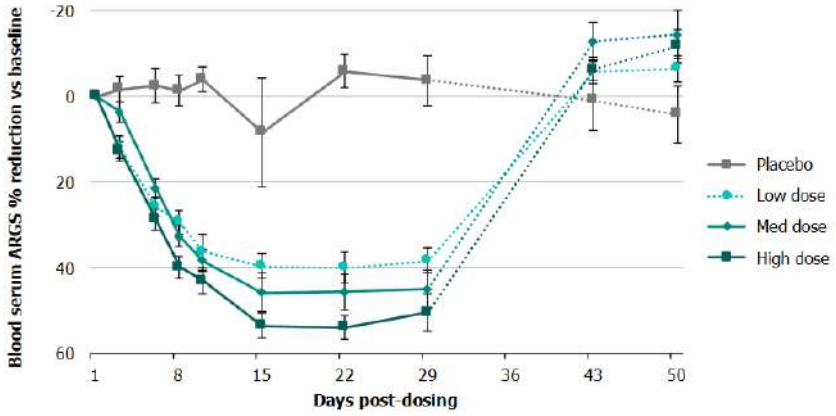
Fig. 22: ADAMTS5 in cartilage destruction



Sources: Ru Bryan et al.

On the back of very promising results seen in a phase Ib data, Servier decided to opt in for GLPG1972 triggering the payment of a EUR6m license fee to Galapagos. Carried out in 30 patients randomised to GLPG1972 at the 100mgQD, 200mgQD or 300mgQD doses vs placebo for 29 days (4 weeks), GLPG1972 showed a significant dose dependent drop in ARGS levels, a marker for target engagement and proxy for cartilage degradation. At the 300mgQD dose, the ARGS level decreased by up to 53% below baseline. In terms of safety, one woman in the high dose group discontinued at day 15 for liver toxicity (ALT increase >x3 ULN) adjudicated to GLPG1792. We note however that this AE proved to be reversible once the treatment stopped.

Fig. 23: GLPG1972 phase Ib study results



Sources: Galapagos.

The ROCCELLA phase II trial was initiated in September 2018 and will recruit 852 patients suffering from osteoarthritis of the knee, including an estimated 300 in the US to be runned by Galapagos. Patients will be randomised to three doses of GLPG1972 (100mgQD, 200mgQD and 300mgQD) vs placebo over a 52-week treatment course at the end of which the reduction in cartilage is to be measured by MRI. Secondary endpoints will be safety and tolerability as well as pain and changes in bone area among others.

In the light of considerable investments that would be necessary in this indication, we assume Galapagos will go to the US market with a partner and derive a 15% market share leading to EUR3.0bn in sales at peak. Osteoarthritis clinical trials have historically yielded very disappointing results, hence our PoS of 30% for this phase II asset. We believe a deal could be inked on the back of positive phase II results in 2020.

Fig. 24: GLPG1972 sales in OA (EURm)

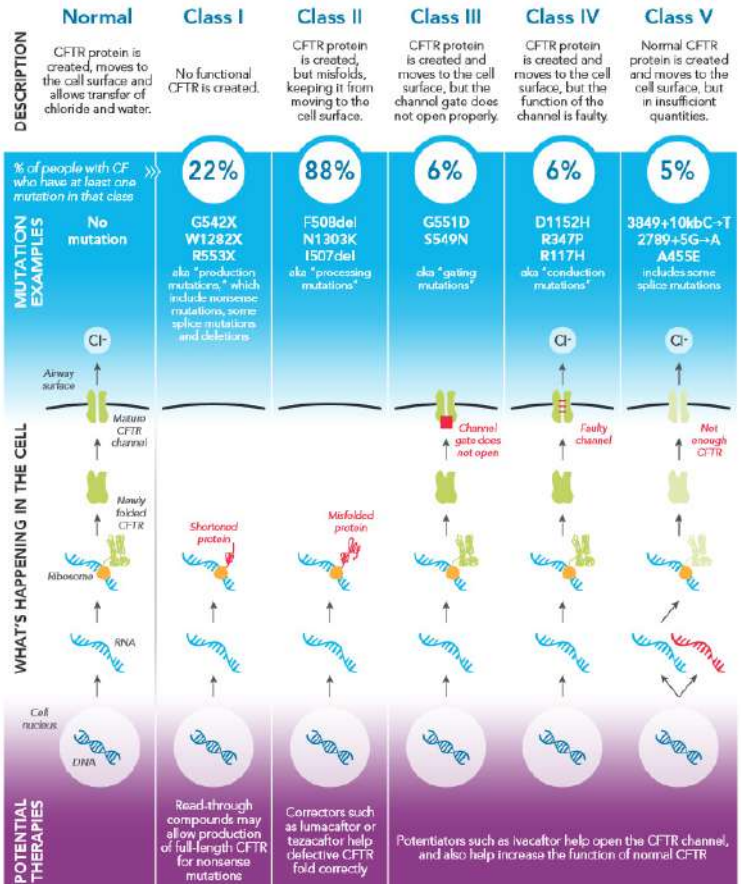
	2023	2024	2025	2026	2027	2028	2029	2030
TOTAL SALES	**15**	**181**	**628**	**1 103**	**1 607**	**2 141**	**2 655**	**3 057**
% growth		**1081%**	**248%**	**76%**	**46%**	**33%**	**24%**	**15%**
EU Sales (m)	15	170	331	500	676	861	1 001	1 005
% growth		1008%	95%	51%	35%	27%	16%	0%
% Sales	100%	94%	53%	45%	42%	40%	38%	33%
o/w Royalties to GLPG	1	14	26	40	54	69	80	80
% royalty rate	8%	8%	8%	8%	8%	8%	8%	8%
US Sales (m)	0	11	297	604	931	1 281	1 654	2 052
% growth			2574%	103%	54%	38%	29%	24%
% Sales		6%	47%	55%	58%	60%	62%	67%

Sources: Bryan, Garnier & Co ests.

Cystic fibrosis is a free upside

Following research advancement in the field of cystic fibrosis (CF) led by Galapagos/AbbVie and Vertex (VRTX US), there is now a consensus that triple combination therapies are to date the most potent drugs in treatment of the underlying cause of this orphan disease with >88% of patients suffering from a Class II mutation (F508del primarily, see chart below). Triple therapies combine correctors and potentiators. 1/ While correctors act by correcting the CFTR protein function, restoring its ability to transport chloride to the epithelial membrane and conduct it through the epithelium, 2/ potentiators help the CFTR protein open up the chloride channel.

Fig. 25: CFTR mutation classes



Sources: Cystic Fibrosis Foundation.

In 2013, Galapagos and AbbVie entered a global CF partnership under which Galapagos received a USD45m upfront. Initially eligible for up to USD360m in milestones plus royalties ranging from the mid-teens to 20%, the milestone package was increased to USD600m in 2016 (royalty rate unchanged). We believe that additional milestones are mainly commercial-related ones, reflecting the broadening of the addressable patient base following clinical progress towards triple combination therapies not included in the deal terms initially.

Galapagos and partner AbbVie have developed several individual potentiators and correctors through multiple clinical trials:

- In the SAPHIRA-1 trial, the efficacy of potentiator GLPG1837 was evaluated in 26 patients harbouring the G551d mutation pre-treated with ivacaftor (Kalydeco). Over a 4-week treatment course, patients were treated at different doses: 125mgBID in week 1, 250mgBID in week 2 and 500mgBID in weeks 3 and 4. After experiencing a 5.6% decline on ppFEV1 after the washout period (73.3% to 69.2%), ppFEV1 returned to the pre-washout period at week 4 (73.1%). The design of the SAPHIRA-2 carried out in patients CF with the S1251N mutation was similar to that of SAPHIRA-1. Kalydeco naïve patients in the SAPHIRA-2 trial reported a maximum ppFEV1 improvement of 5% at week 4, a touch lower compared to a >9% ppFEV1 improvement plateau effect reported by ivacaftor as early as week 4.
  In all, the results from these two trials were not differentiated enough from ivacaftor and Galapagos/AbbVie decided to pursue the development of another potentiator, GLPG2451 which yielded better results and was already being studied in a preclinical trial in combination with corrector GLPG2222.

- The ALBATROSS and FLAMINGO phase Ib trials evaluated corrector GLPG2222 in F508del heterozygous and homozygous CF patients respectively.

  The ALBATROSS trial aimed to assess the efficacy of GLPG2222 on top of ivacaftor (potentiator) in 37 patients, randomised to GLPG2222 at the 150 or 300mgQD dose vs placebo. After a 4-week treatment course at the highest dose, patients showed a 2.2% ppFEV1 increase compared with a -0.8% decline in the placebo and a statistically significant decrease in sweat chloride of -6mmol/L ($p<0.05$). Although evaluating the drug as a monotherapy, these results compared well with Vertex Symdeko (tezacaftor + ivacaftor / ivacaftor) on sweat chloride change (-6.4mmol/L sweat chloride decline at 4 weeks, Donaldson SH et al). AEs were well balanced between the active and placebo groups with only one patient in the GLPG2222 150mgQD group experiencing an SAE (headache on day-2) but did not discontinue the study. Only two patients discontinued the trial for reasons not linked to GLPG2222 (one patient stopped attending study visits and one was wrongly dosed).
  In the FLAMINGO phase Ib trial, 59 patients were recruited and randomised to four different doses of once daily GLPG2222 as a monotherapy (50, 100, 200 and 400mgQD) after a 4-week washout period, which did not impact the length of recruitment, closed within five months. At week 4, the 200mgQD dose showed a statistically significant -18.3mmol/L decrease in sweat chloride ($p=0.0001$). However, no dose showed impact of ppFEV1. AEs were well balanced between the active and placebo groups. SAEs were not dose dependent with one patient experiencing two pulmonary exacerbations in the dose 2 group compared with two patients experiencing the same AE in the placebo group.

Despite positive progress with potentiator GLPG2451 and corrector C1 GLPG2222, the recent underwhelming development of C2 corrector in the PELICAN trial due to which the partnership was put under review, as well as low expectations ahead of the interim read-out of the FALCON trial, all warrant caution.

- Mixed results from the PELICAN trial. This phase II trial enrolled 28 F508del homozygous patients randomised to Galapagos' C2 corrector GLPG2737 (n=14) on top of Orkambi (lumacaftor+ivacaftor) vs placebo (n=8) for 4 weeks. While a significant sweat chloride decrease of 19.6mmo/L vs placebo (p=0.02), change from baseline in ppFEV1 vs placebo of 3.4% (p=0.08) fell short of expectations. Indeed, Vertex correctors VX-445 and VX-659 reported ppFEV1 improvements of 11% (p<0.0001) and 9.7% (p<0.0001) when added to the combination of tezacaftor (corrector) and ivacaftor (potentiator) in the same F508del homozygous patient population. Vertex completed the recruitment of the VX-659 phase III trial in September and should complete that for VX-445 phase III trial by the end of the year. By mid-2019, its first triple combination should be filed with the FDA.

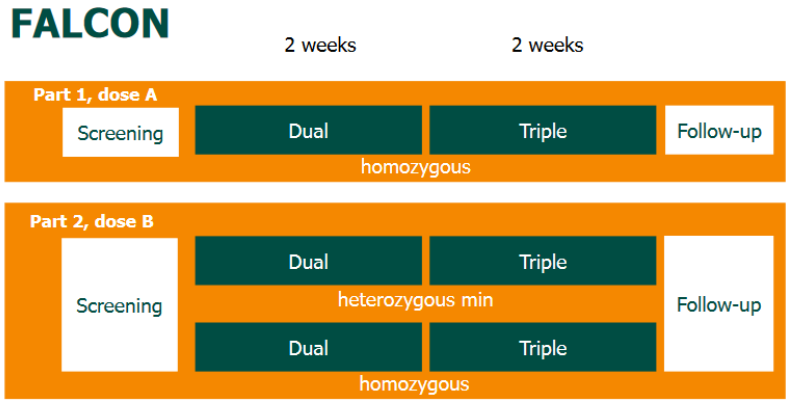
In view of the disappointing results from the PELICAN trial, AbbVie decided not to proceed with a second triple combination consisting of correctors GLPG2222+GLPG2737 and potentiator GLPG3067. We believe this is a clear sign of AbbVie deprioritising the programme following accumulated delays vis-a-vis Vertex, which has set a high bar for triple combinations and has leading commercial operations. The Galapagos share price took a hit on this announcement and we have decided not to include the CF programme in our valuation, preferring to view it as free upside. This cautious stance is all the more justified by the low expectations we have for the interim read-out of the FALCON trial, expected in coming weeks (i.e. early Q4 2018).

- The first part of the FALCON trial evaluates the efficacy and safety of GLPG2222+GLPG2451 dual combination in 10 F508del homozygous CF patients for two weeks, at the end of which corrector GLPG2737 is to be added for an extra two weeks.

We do not expect the first part of this trial to show a significant improvement in ppFEV1 as GLPG2737 will not have reached maximum exposure level after only two weeks of treatment.

- The second part of the FALCON study will evaluate two cohorts of F508del homozygous (n=8) or F508del heterozygous (n=8) patients. These two cohorts will receive a higher dose of the GLPG222+GLPG2451 dual combination for two weeks at the end of which the triple combination of GLPG2222+GLPG2451+GLPG2737 will be administered for an additional two weeks. Final results are to be reported in late Q1 2019/early Q2 2019.

Fig. 26: FALCON trial design



Sources: Galapagos.

In all, we see the interim results of the FALCON trial and the subsequent outcome of the partnership review as free upside. We would integrate the CF programme into our estimates once the outcome of the partnership review is known, bearing in mind that Galapagos will hold its R&D day on 25th October. This event could provide an opportunity for the group to communicate on a clear strategy (positioning) for the CF franchise if the partnership review has been concluded by then

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Price Chart and Rating History

Galapagos

Ratings		
Date	**Ratings**	**Price**
04/01/12	BUY	EUR10.55

Target Price	
Date	**Target price**
27/10/17	Under review
05/01/17	EUR67
02/05/16	EUR64
06/04/16	EUR62
26/01/16	EUR63
17/12/15	EUR64
01/10/15	EUR52
28/09/15	EUR50
11/08/15	EUR61
30/07/15	EUR57
08/06/15	EUR51
28/04/15	EUR41.5
15/04/15	EUR32.5
14/04/15	EUR28.5
17/03/15	EUR26

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NEUTRAL	Opinion recommending not to trade in a stock short-term, neither as a BUYER or a SELLER, due to a specific set of factors. This view is intended to be temporary. It may reflect different situations, but in particular those where a fair value shows no significant potential or where an upcoming binary event constitutes a high-risk that is difficult to quantify. Every subsequent published update on the stock will feature an introduction outlining the key reasons behind the opinion.
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15 St. Botolph Street	75008 Paris	80538 Munich	8001 Zurich	16th floor
London EC3A 7BB	France	Germany	Switzerland	New York, NY 10022
United Kingdom	+33 1 56 68 75 20	+49 89 2422 62 11	+41 44 991 3300	United States
+44 207 332 2500				+1 212 337 7000

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