Degroof Petercam

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BUY

Price (18/07/2018)	EUR 87.92
Target price	110.00
Risk	High
Reuters	GLPG AS
Bloomberg	GLPG NA
Shares number (m)	51.23
Market cap. (m)	4,505
Cash Position 12/18e (m)	892
1 year price perf.	31.6%
Diff. with Euro Stoxx	29.3%
Volume (sh./day)	435,022
H/L 1 year	97.24 - 62.63
Free Float	76.8%
Gilead	13.2%
Van Herk Investments	10.0%

Company description

Galapagos is a biotech company focused mainly on small molecules in inflammatory and fibrotic indications. The company has a broad and mature pipeline, its lead product is in Ph3. In addition Galapagos is supported by strong partnerships.

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Galapagos

CrackerJAK

Best-in-class potential

- Our interactions with key opinion leaders in the field taught us that targeted small molecule inhibitors are expected to transform the treatment paradigm in the field of autoimmune disease, with filgotinib potentially being a best-in-class JAK inhibitor.
- Following the commercially available Xeljanz and Olumiant, the next wave of JAK inhibitors, upadacitinib and filgotinib, have been developed to be more selective for JAK1 and thus to show a superior safety profile. To date, filgotinib has been able to show the most favourable data concerning adverse events that characterize this class of therapies.
- In 2H18, Galapagos and partner Gilead aim to report topline results of the Phase III FINCH-2 study in rheumatoid arthritis. Gilead progressed fast through recruitment, bringing ahead the readout of FINCH-1 and 3 to 1H19.

Multiple shots at goal

- Besides the progress with filgotinib, the past year yielded a wealth of results with remarkable data from Galapagos' proprietary idiopathic pulmonary fibrosis program, which moved into Phase III, as well as from the atopic dermatitis and osteoarthritis trials.
- On the other hand, the cystic fibrosis program experienced a significant setback when the PELICAN trial failed to show a strong effect of '2737 on top of Orkambi. As this component is part of the first triple combination in the clinic, the chances of success for Galapagos in CF are becoming slim.

Lead asset nearing Phase III readout

- The first Phase III results in rheumatoid arthritis will definitely move the needle for Galapagos. The FINCH-2 study targets the most difficult to treat RA patients. Positive results will thus further de-risk the larger FINCH-1/3 studies.
- The upcoming news flow will provide multiple value inflection points through which significant upside could materialize in the coming quarters.
- Incorporating feedback from the medical community, we have taken a more bullish stance on the future of JAK inhibitors in the field of autoimmune disease in general and the best-in-class potential of filgotinib specifically. Adjusting our assumptions to reflect this item, we raise our TP to EUR 110 from EUR 99.

EUR	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
Revenues	90.0	60.6	151.6	155.9	183.8	151.3	146.2
R & D	111	130	140	219	284	312	328
EBIT	-36.6	-89.4	-11.5	-89.8	-133.6	-197.6	-223.3
Decl. profit	30.2	-118	54.0	-116	-139	-205	-232
EPS	1.02	-3.03	1.17	-2.27	-2.71	-4.00	-4.53
EV/Revenues	2.9	30.9	12.1	18.4	19.7	25.3	27.9
EV/R & D	2.4	14.4	13.2	13.1	12.7	12.3	12.4
P/E	15.2	nm	52.2	nm	nm	nm	nm
Net Cash	198	347	980	1,151	892	676	422

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ACR: American College of Rheumatology; **AS**: ankylosing spondylitis; **ASAS**: assessment in ankylosing spondylitis; **ASDAS**: ankylosing spondylitis disease activity score; **AtD**: atopic dermatitis; **bDMARD**: biological DMARD; **CD**: Crohn's disease; **CF**: cystic fibrosis; **csDMARD**: conventional synthetic DMARD; **DMARD**: disease-modifying anti-rheumatic drug; **EULAR**: European League Against Rheumatism; **FDA**: Food and Drug Administration; **IBD**: inflammatory bowel disease; **IPF**: idiopathic pulmonary fibrosis; **IR**: inadequate responder; **JAK**: Janus kinase; **KOL**: key opinion leader; **mAb**: monoclonal antibody; **MTX**: methotrexate; **NK cell**: natural killer cell; **NSAID**: non-steroidal anti-inflammatory drug; **OA**: osteoarthritis; **ppFVC**: percent predicted forced vital capacity; **PsA**: psoriatic arthritis; **RA**: rheumatoid arthritis; **UC**: ulcerative colitis.

Summary

The EULAR 2018 conference focused strongly on the emerging JAK inhibitors in inflammatory diseases. Several KOLs indicated that targeted small molecules are making their way into the 'treat-to-target' strategy of physicians and are expected to take up a significant part of the biologics market in the future.

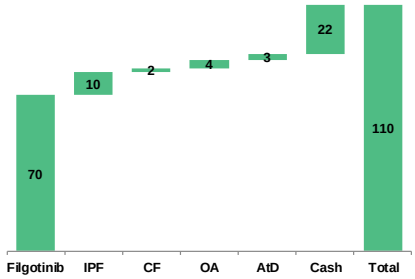
Two JAK inhibitors have received marketing approval to date, Xeljanz (Pfizer) and more recently Olumiant (Eli Lilly). The safety profile of these molecules has been severely scrutinized, yet Pfizer was able to report over EUR 1.3bn in sales for Xeljanz in 2017 and Olumiant showed the most successful launch to date in rheumatoid arthritis. The next wave of JAK inhibitors, upadacitinib and filgotinib, are designed to be more selective for JAK1 and are consequently expected to present an improved safety profile. Long-term data presented at the EULAR conference confirmed filgotinib's sustained efficacy and safety profile. In our view, filgotinib has the potential to become the best-in-class JAK inhibitor. In this report, we provide our view on the data obtained with filgotinib in comparison to its competitors. Incorporating feedback from the medical community, we have taken a more bullish stance on the future of JAK inhibitors in the field of autoimmune disease in general and the best-in-class potential of filgotinib specifically. As a result, filgotinib represents EUR 70 per share in our sum-of-the-parts valuation.

Galapagos has demonstrated the strength of its technology platform over the past year by delivering strong results in several inflammatory and fibrotic diseases through new mechanisms of action. GLPG1690 has moved fast through clinical development, now in a pivotal Phase III program in idiopathic pulmonary fibrosis. This life-threatening orphan indication is in dire need of disease-modifying treatments. In cystic fibrosis, Galapagos and AbbVie initiated the triple combination program, FALCON, for which first results will be announced in 3Q18. With Vertex reporting strong efficacy results, the bar has been set high. Furthermore, the disappointment of the C2 corrector in the PELICAN trial has significantly decreased the chances of success of Galapagos' triple combination in our view. These evolutions led us to decrease the value of the CF program from EUR 6 to EUR 2 per share.

The programs in atopic dermatitis and osteoarthritis have remained somewhat under the radar, but these large markets could also represent a significant opportunity.

The main focus point this year will clearly be Galapagos and Gilead's JAK inhibitor, filgotinib. The Phase III studies in rheumatoid arthritis (RA) are nearing completion and the results from additional proof-of-concept studies in other inflammatory indications will be announced in the coming months. The upcoming news flow will provide multiple value inflection points through which significant upside could materialize in the coming quarters. Our sum-of-the-parts valuation points to a rNPV of EUR 5.6bn or EUR 110/share.

Exhibit 1 Sum-of-the-parts valuation (EUR/share)



Source: Degroof Petercam estimates; CF: cystic fibrosis; IPF: idiopathic pulmonary fibrosis (GLPG1690); OA: osteoarthritis (GLPG1972); AtD: atopic dermatitis (MOR106)

Exhibit 2 News flow

	2H18	1H19
Filgotinib	Ph III Rheumatoid arthritis - FINCH-2 Ph II Ankylosing spondylitis - TORTUGA	Ph III Rheumatoid arthritis - FINCH-1 & 3 Ph II Cutaneous lupus erythematosus Ph II Sjögren's syndrome
IPF	Ph III start dosing - ISABELA	
CF	Ph II triple combination - FALCON part 1	[Initiation and data additional triple combinations]
AtD	Ph II ongoing - IGUANA	
OA	Ph II start dosing - ROCELLA	

Source: Galapagos

The era of targeted small molecules in autoimmune diseases

As confirmed by the focus points at the **EULAR (European League Against Rheumatism) conference** this year, the **era of the targeted small molecules** is arriving in autoimmune diseases. Increasing evidence is being built on the benefit of JAK inhibitors in rheumatoid arthritis as well as in other indications. To this day, an important unmet need exists in autoimmune disease. For example, many patients with RA still fail to reach treatment targets and rates of sustained clinical remission remain relatively modest (5-45%). Furthermore, secondary loss of response is a common feature of currently available therapies. Additionally, patients consider the route of administration the most important attribute of their treatment, with 56.4% preferring oral administration[1].

In the targeted small molecule field, two JAK inhibitors are already on the market today. Tofacitinib (Xeljanz; Pfizer), a pan-JAK inhibitor, was approved by the FDA in 2012 and is indicated for the treatment of RA, psoriatic arthritis and ulcerative colitis. Despite being associated with severe adverse events, **Xeljanz reached USD 1.3bn sales in 2017**. In April this year, baricitinib (Olumiant; Eli Lilly) was evaluated by the FDA's arthritis advisory committee. This was an important event in the JAK inhibitor field, giving insight into the focus points of the committee. Eli Lilly already filed for approval last year, but this was rejected as the FDA demanded additional data on safety and dosing. In the beginning of June, Eli Lilly and partner Incyte finally secured FDA approval for their JAK inhibitor in rheumatoid arthritis. Only the lower dose was approved and this with a black box safety warning (serious infections, malignancy and thrombosis risks).

Two other JAK inhibitors are finalising Phase III trials, upadacitinib (AbbVie) and filgotinib. These JAK inhibitors have been developed to be more selective for JAK1 as compared to the tofacitinib (JAK1/JAK2/JAK3) and baricitinib (JAK1/JAK2), aiming to **improve the safety profile**. Filgotinib has shown a strong safety profile in Phase II studies. Recent 108 week data again underpinned these observations. We await confirmation of the data from the Phase III trials for which we expect results in 2H18/1H19.

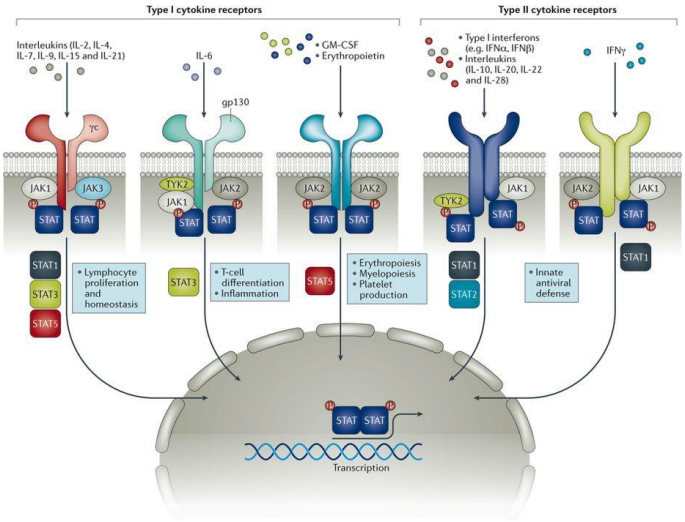
The concept of JAK selectivity – implications on safety profile

In contrast to common biologicals, JAK inhibitors target intracellular components of the inflammatory cascade and as such affect a range of immunological factors.

JAK inhibitors are **never solely specific for a certain JAK enzyme**, however they may have a higher affinity for one JAK over the other. Therefore not all JAK inhibitors will have the same biologic effect. As is the case with biological therapy, JAK inhibition can lead to serious and opportunistic infections. The aim today is to **target JAK1 without hitting JAK2 and JAK3** too much in order to avoid negative effects such as anaemia (JAK2) or decreases in NK cells (JAK3).

[1] Alten R, et al. Patient Pref Adherence 2016

Exhibit 3 JAK signalling pathway



Source: Winthrop KL, et al. Nature Review Rheumatology 2017

Difference in laboratory parameters can't be solely explained by differential activity of JAK inhibitors against JAK2 or JAK3. Clinical exposure to JAK inhibitors is **dependent on dose and time** and also the inhibition of signalling downstream (cytokine levels) has been shown to be time dependent[2]. During clinical development, the aim is to optimize the dose to give maximum efficacy with minimum toxicity and as such define a '**therapeutic window**'. Pharmacology indeed plays an important role as we see for example upadacitinib (JAK1 selective) clearly affecting (decreasing) haemoglobin levels at higher doses (30 mg), but less at lower doses (15 mg), while efficacy results are comparable for both doses.

Exhibit 4 Potency and in vitro cytokine inhibition

	Enzyme assay IC_{50} (nM)				Cellular assay IC_{50} (nM)		
Compound	**JAK1**	**JAK2**	**JAK3**	**TYK2**	**IL-6**	**IL-15**	**EPO**
Tofacitinib	15.1	77.4	55.0	489.0	75.4	55.8	302.0
Baricitinib	4.0	6.6	787.0	61.0	21.1	259.0	87.8
Upadacitinib	47	120	2,304	4,690	11	22	649
Filgotinib	363	2,400	>10,000	2,600	918	2,140	13,200

Source: Clarke JD, et al. J Med Chem 2014; Parmentier JM, et al. BMC Rheum (In Press)

Exhibit 5 Overview safety profile JAK inhibitors

	Tofacitinib	Baricitinib	Upadacitinib	Filgotinib
Lymphocyte number	↓	No change	↓	No change
NK cell number	↓	↓	↓	No change
Neutrophil number	↓	↓	↓	↓
Haemoglobin level	↑	↓	↓	↑
Platelet count	↓	No change	-	↓
Liver transaminase level	↑	↑	↑	No change
Creatine phosphokinase level	↑	↑	↑	-
HDL level	↑	↑	↑	↑
LDL level	↑	↑	↑	No change
Creatine level	↑	↑	↑	↑

Source: Winthrop, Nature Reviews Rheumatology 2017

The decrease in **haemoglobin** by upadacitinib points to an effect on JAK2 and consequently erythropoietin (EPO), a factor involved in the production of red blood cells. It is an important parameter, as RA patients already display anaemia and may feel significantly better when haemoglobin levels rise. RA patients often also display **platelet count** elevation. A further increase in platelets due to JAK inhibitor treatment could be associated with an increased risk for thrombotic events. Another important parameter to the safety profile is the **NK cell** number. NK cells are an important defence in the innate immune system, lower numbers may lead to increased infection and malignancy rates.

[2] Dowty ME, et al. Clin Exp Rheumatol 2016

Exhibit 6 **Overview treatment-emergent adverse events JAK inhibitors**

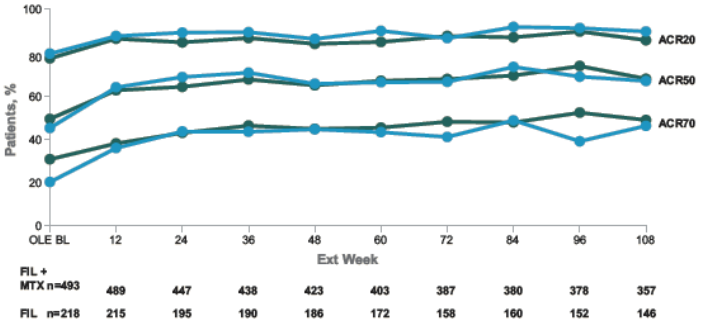
Cases per 100 PYE	Tofacitinib	Baricitinib	Upadacitinib	Filgotinib
PYE	19,406	6,637	725	1,708
Serious infection	2.7	2.9	2.3	1.5
Herpes Zoster	3.9	3.2	3.7	1.2
DVT/PE	0.2*	0.5	0.7	0.1

Source: Cohen SB, et al. Ann Rheum Dis 2017; Genovese MC, et al. ACR 2017 ABSTRACT 511; Genovese MC, et al. EULAR 2018 ABSTRACT SAT0236; *based on 5,891 patient years; PYE: patient years exposure; DVT: deep vein thrombosis; PE: pulmonary embolism

Galapagos and Gilead presented new data on the long term safety and efficacy of filgotinib in rheumatoid arthritis patients (DARWIN3 study) at the EULAR conference in June[3]. The **week 108 data indicate a safety profile consistent with previously reported data**. There was no difference in safety profile in patients receiving filgotinib 200 mg QD or 100 mg BID with or without methotrexate.

Importantly, the **efficacy results were sustained** in patients who remained on treatment, regardless of dosing regimen or background methotrexate treatment. ACR20/ACR50/ACR70 responses were achieved in 87%, 68% and 48% of patients, respectively and 72% achieved DAS28-CRP ≤ 3.2.

Exhibit 7 **ACR responses were sustained up to week 108**



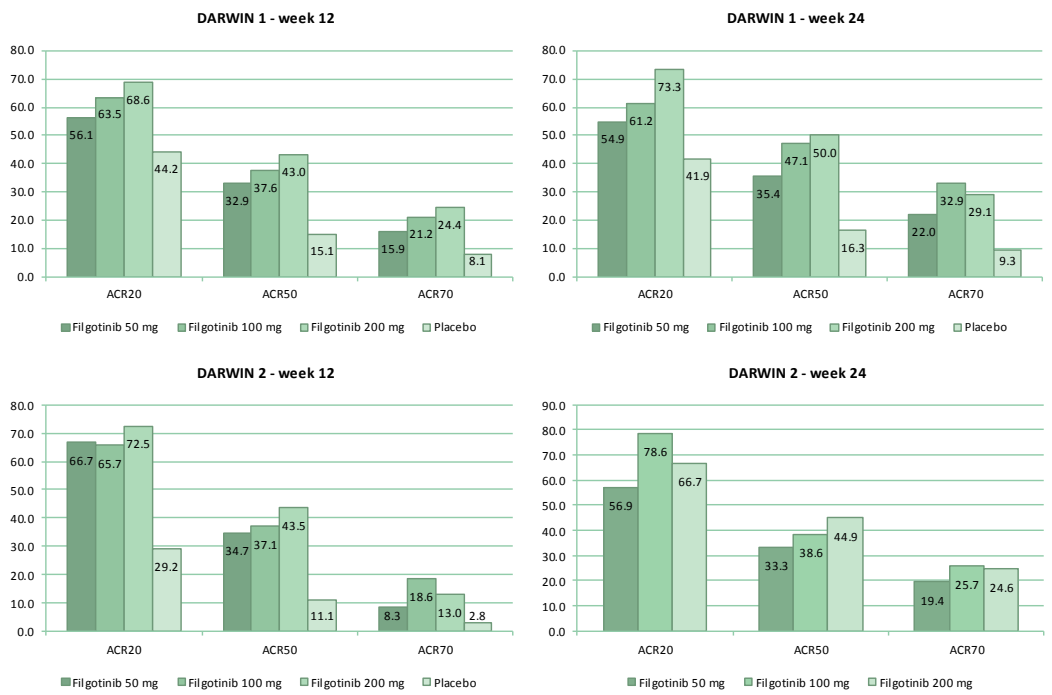
Source: Westhovens R, et al. EULAR 2018 ABSTRACT SAT0200

First Phase III readout in rheumatoid arthritis nearing

Galapagos showed strong results in its Phase IIb studies with filgotinib in biologic naïve RA patients (DARWIN 1 and 2). The DARWIN1 study evaluated filgotinib in patients with moderately to severe active RA receiving a stable dose of methotrexate, while the DARWIN2 study included a 4-week washout from methotrexate. Interestingly, data from both trials indicate that filgotinib as monotherapy shows comparable efficacy results to filgotinib as add-on in combination with methotrexate.

[3] EULAR 2018 poster 0200 – Long-term safety of filgotinib in the treatment of rheumatoid arthritis: week 108 data from a Phase IIb open-label extension study

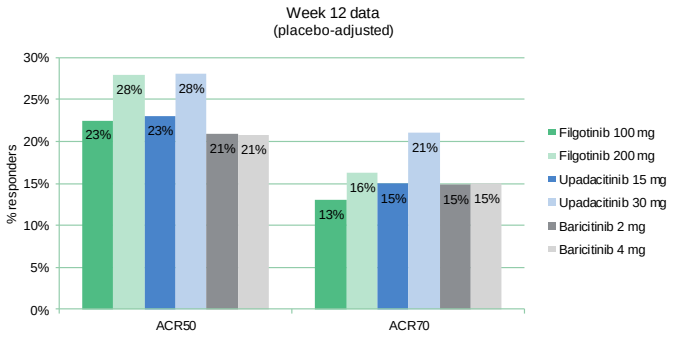
Exhibit 8 DARWIN 1 and DARWIN 2 reported ACR responses



Source: Westhovens R, et al. Ann Rheum Dis 2017; Kavanaugh A, et al. Ann Rheum Dis 2016

Efficacy results of the filgotinib Phase IIb studies compare positively to the results of other JAK inhibitors (tofacitinib, baricitinib and upadacitinib).

Exhibit 9 Comparison of ACR50 and ACR70 responses



Source: Westhovens R, et al. Ann Rheum Dis 2017; Burmester GR, et al. Lancet 2018; Dougados M, et al. Ann Rheum Dis 2017

Gilead communicated the completion of recruitment in the FINCH studies in May 2018. Consequently we can expect **topline results of the FINCH-2 study around September** this year (we expect Gilead to present the data set at the next ACR conference, 19th-24th October 2018).

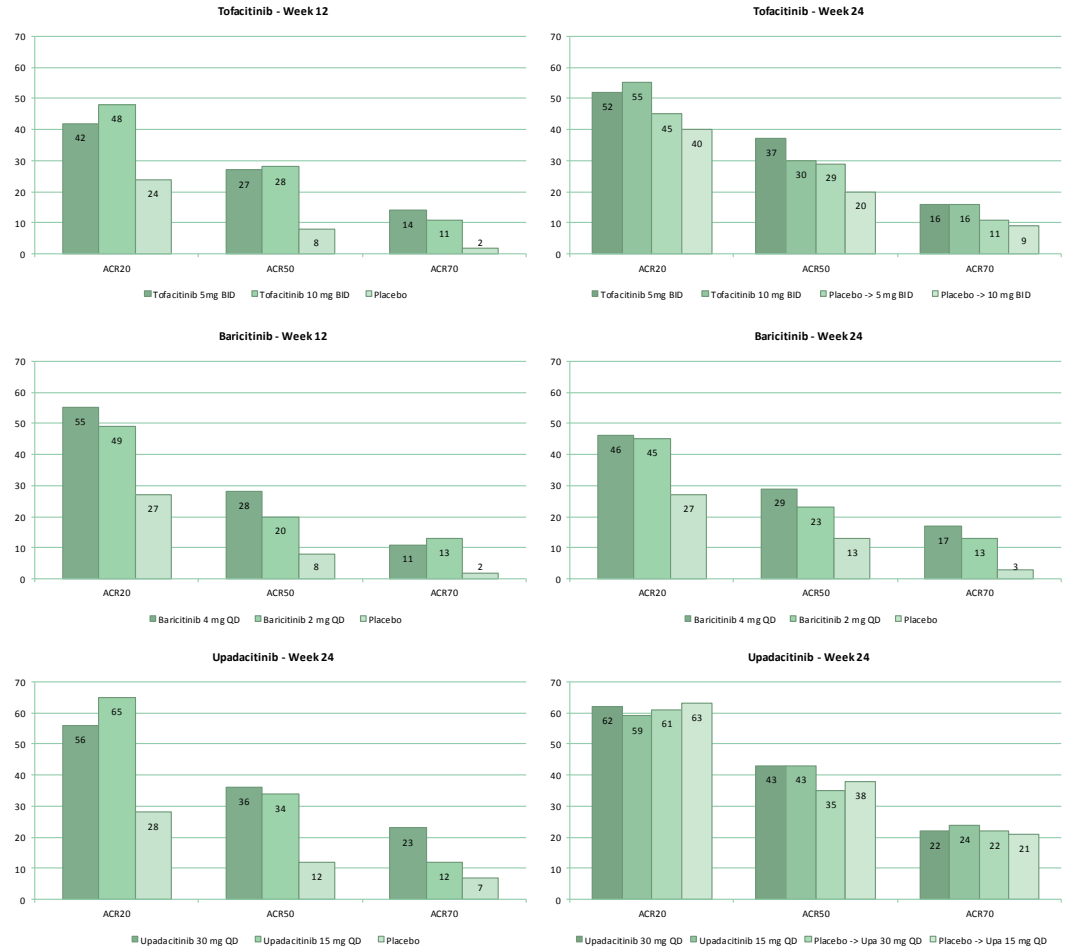
Exhibit 10 Phase III rheumatoid arthritis studies

	N° patients	Population	Cohorts	Treatment duration	Primary endpoint
FINCH-1	1,650	Insufficient response to methotrexate	- Filgotinib 100mg + MTX - Filgotinib 200 mg + MTX - Adalimumab + MTX - Placebo + MTX	52 weeks	ACR20 at week 12
FINCH-2	423	Insufficient response to biologic treatment	- Filgotinib 100mg + DMARD - Filgotinib 200 mg + DMARD - Placebo + DMARD	24 weeks	ACR20 at week 12
FINCH-3	1,200	Naïve to methotrexate	- Filgotinib 100 mg + MTX - Filgotinib 200 mg + MTX - Filgotinib 100 mg - MTX	52 weeks	ACR20 at week 24

Source: Galapagos

The FINCH-2 study targets patients with an insufficient response to biologic treatments (bDMARD-IR), as such this is the most difficult to treat patient population. Other JAK inhibitors have demonstrated compelling efficacy results in this population. We will use these results as a benchmark to compare the FINCH-2 results, keeping in mind that study populations, baseline characteristics, etc. may vary and direct comparisons should be made carefully.

Exhibit 11 ACR responses in bDMARD-IR patients



Source: Burmester GR, et al. Lancet 2013 ; Genovese MC, et al. N Engl J Med 2016; Genovese MC, et al. ACR 2017 Abstract 10L; Genovese MC, et al. EULAR 2018 Abstract SAT0219; Genovese MC, et al. Lancet 2018

EULAR treatment recommendations currently indicate the use of biologics in combination with csDMARDs such as methotrexate. All bDMARDs have superior efficacy when combined with methotrexate compared to methotrexate alone. However, we learned from our interactions with KOLs that physicians and patients are looking for treatments that do not require the combination of methotrexate. Indeed, in clinical practice up to 30% of patients receive biologics as monotherapy as a result of the significant adverse events related to csDMARDs, potential intolerance and desire to reduce medication. As such, we will be looking closely into the monotherapy results.

The FINCH-1 and 3 studies are expected to deliver topline results in 1H19. These studies will evaluate the potential of filgotinib in combination with methotrexate or as monotherapy in patients with an inadequate response or naïve to csDMARDs.

Opportunity in inflammatory bowel disease

Crohn's disease (CD) and ulcerative colitis (UC), two types of inflammatory bowel disease (IBD), are severe indications affecting the digestive system. CD can affect any part of the gastrointestinal tract, but most often involves the large and small intestines, while UC is localized to the large intestine or colon. For both indications, the exact cause is unknown but likely involves a combination of environmental, immune as well as genetic factors. Symptoms of IBD include diarrhoea, fever and fatigue, abdominal pain and cramping, blood in the stool, reduced appetite and unintended weight loss.

IBD prevalence has been reported to be rising significantly over the past decades, today over 3 million adults in the US are living with IBD. In 2017, the global market was estimated at USD 6.7bn, with biologic therapies accounting for 57% of market share.

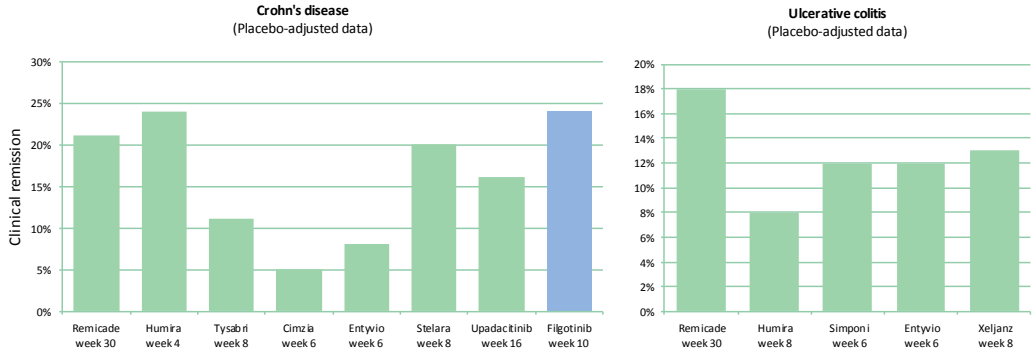
Guidelines for the treatment of IBD recommend the use of anti-TNF agents in moderate-to-severe IBD if non-biological therapy fails. However, these therapies are not effective in all patients and patients that do respond initially may lose responsiveness over time. More specifically, up to 50% of patients experience secondary loss of responses as reported in clinical trials.

The only JAK inhibitor on the market in IBD to date is tofacitinib, approved for the treatment of ulcerative colitis (May 2018). Clinical development of tofacitinib in Crohn's disease was discontinued after the readout of a Phase IIb trial that could not indicate statistical significance over placebo.

Galapagos ran a Phase II placebo-controlled trial (FITZROY) with filgotinib in patients with moderate-to-severe Crohn's disease. The study enrolled 174 patients of which 130 were randomized into the treatment arm (200 mg filgotinib) and 44 in the placebo arm. In the ITT population, 60 of 128 (47%) patients treated with filgotinib achieved clinical remission (defined as CDAI<150) at week 10 versus 10 of 44 (23%) treated with placebo (p=0.0077).

Among anti-TNF naïve patients, 60% in the filgotinib group achieved clinical remission, compared to 13% in the placebo group. Among anti-TNF experienced patients, 37% of patients achieved clinical remission with filgotinib versus 29% in the placebo group.

Exhibit 12 Overview of efficacy results of biologic treatments in CD and UC



Source: FDA drug database; Comparisons should be made with caution as patient populations, time points of assessment, etc. vary between trials

Galapagos and Gilead are now running Phase III trials with filgotinib in Crohn's disease and ulcerative colitis. An independent Data Monitoring Committee recently recommended the Phase IIb study in ulcerative colitis to proceed as planned into Phase III at both the 100 mg and 200 mg doses, no detailed results were published.

Targeting a broad label

In 2017, Gilead and Galapagos initiated several additional proof-of-concept studies in various inflammatory indications with the aim to broaden the future label of filgotinib.

Exhibit 13 Overview of ongoing filgotinib Phase II proof-of-concept studies and expected completion

Indication	Trial description	Expected completion
Psoriatic arthritis	A randomized, double-blind, placebo-controlled, multicenter, Phase II study to assess the efficacy and safety of filgotinib administered for 16 weeks to subjects with moderately to severely active psoriatic arthritis	March 2018 Data reported 2Q18
Ankylosing spondylitis	A randomized, double-blind, placebo-controlled, multicenter, Phase II study to assess the efficacy and safety of filgotinib administered for 12 weeks to subjects with active ankylosing spondylitis	June 2018 Results expected 3Q18
Cutaneous lupus erythematosus	A Phase 2, randomized, double-blind, placebo-controlled study to assess the safety and efficacy of filgotinib and GS-9876 in female subjects with moderately-to-severely active cutaneous lupus erythematosus (CLE)	October 2018 Results expected 1H19
Sjögren's syndrome	A randomized, Phase 2, double-blind, placebo-controlled study to assess the safety and efficacy of filgotinib, GS-9876 and GS-4059 in adult subjects with active Sjogren's syndrome	March 2019 Results expected 1H19
Small bowel Crohn's disease	A Phase 2, double-blind, randomized, placebo-controlled study evaluating the efficacy and safety of filgotinib in the treatment of small bowel Crohn's disease (SBCD)	March 2020
Fistulizing Crohn's disease	Efficacy and safety of filgotinib in the treatment of perianal fistulizing Crohn's disease	April 2020
Lupus nephropathy	A Phase 2, randomized, double-blind, multicenter study evaluating the safety and efficacy of filgotinib and GS-9876 in subjects with lupus membranous nephropathy (LMN)	July 2020
Uveitis	A Phase 2, randomized, placebo-controlled trial evaluating the efficacy and safety of filgotinib in subjects with active noninfectious uveitis	December 2020

Source: Galapagos

The results of the Phase II proof-of-concept study in psoriatic arthritis were recently announced, while the study in ankylosing spondylitis will read out in 3Q18.

Positioning of JAK inhibitors in psoriatic arthritis

Psoriatic arthritis is a type of arthritis affecting 5-30% of patients with psoriasis. Over 1 million people in the US and Europe are diagnosed with the disease. Treatment regimens include the use of NSAIDs and DMARDs.

The Phase II proof-of-concept study for filgotinib in psoriatic arthritis included patients on stable DMARDs, with 85% of patients naïve to anti-TNF therapy. The placebo-controlled study assessed the safety and efficacy of filgotinib in 131 patients. The primary endpoint was the percentage of patients reaching ACR20 response as compared to placebo at week 16.

The primary endpoint of ACR20 at week 16 was achieved. There was an **ACR20 response of 80% for filgotinib** as compared to 33% for placebo (p<0.001). However, to physicians in the clinical setting, the ACR20 score is less important; ACR50 is clinically more relevant. The ACR50 and ACR70 scores were also significantly higher for filgotinib versus placebo (ACR50: 48% with filgotinib vs 15% placebo; ACR70: 23% with filgotinib vs 6% placebo). The table below indicates the competitiveness of filgotinib in the treatment of psoriatic arthritis.

Exhibit 14 Overview of efficacy results of approved biologicals for psoriatic arthritis

		ACR20		ACR50		ACR70	
		Treatment	Placebo	Treatment	Placebo	Treatment	Placebo
Adalimumab (Humira) *(40 mg SQ every 2 weeks)*	12 wks	58%	14%	36%	4%	20%	1%
Secukinumab (Cosentyx) *(300 mg SQ monthly)*	16 wks	57%	18%	35%	6%	15%	2%
Ustekinumab (Stelara) *(45 mg SQ every 12 weeks)*	12 wks	41%	21%	25%	9%	12%	2%
Ixekizumab (Taltz) *(80 mg SQ every 4 weeks)*	24 wks	58%	30%	40%	15%	23%	6%
Apremilast (Otezla) *(30 mg oral tablet bid)*	16 wks	38%	19%	16%	6%	4%	1%
Tofacitinib (Xeljanz) *(5 mg oral tablet bid)*	12 wks	50%	33%	28%	10%	17%	5%
Filgotinib* *(200mg oral tablet qd)*	16 wks	80%	33%	48%	15%	23%	6%

Source: FDA drug database; Galapagos press release May 30, 2018

Ankylosing spondylitis

The second proof-of-concept study with filgotinib to read out this year will be the trial in ankylosing spondylitis, a chronic disease that involves inflammation of the spine and other areas of the body. About 2 million people suffer from this disease in the US, Europe and Japan.

Galapagos' Phase II placebo-controlled study will enrol 116 patients. The primary endpoint, measured at week 12, will be the ankylosing spondylitis disease activity score (ASDAS) in filgotinib treated patients compared to placebo. This endpoint relates most to disease activity, as opposed to ASAS scores that are more related to the patient.

The table below shows the efficacy results of biologicals approved or in development for the treatment of ankylosing spondylitis. Note that AbbVie is also evaluating its JAK inhibitor, upadacitinib, in a Phase II placebo-controlled trial with primary completion in December 2018.

Exhibit 15 Overview of efficacy results of biologic treatments in ankylosing spondylitis

		ASAS20		ASDAS	
		Treatment	Placebo	Treatment	Placebo
Adalimumab (Humira) *(40 mg SC every 2 weeks)*	12 wks	58%	21%	-	-
Secukinumab (Cosentyx) *(150 mg SC monthly)*	16 wks	61%	28%	-	-
Tofacitinib (Xeljanz)* *(5 mg oral tablet bid)*	12 wks	80%	41%	64%	28%

Source: FDA drug database; van der Heijde D, et al. Ann Rheum Dis 2017 *not approved

Market opportunity and valuation of filgotinib

The inflammatory disease market is substantial and continues to grow (CAGR 4% expected for the next 10 years). Within this field, rheumatoid arthritis drugs represent the largest segment of about 40% or over USD 20bn.

Our valuation model for filgotinib includes the following assumptions:

- Rheumatoid arthritis: It is estimated that about 50% of patients with moderate-to-severe RA do not adequately respond to csDMARDs (such as methotrexate). Today, the majority of patients in this group are subsequently treated with anti-TNFs, while the minority receives a different biological therapy. We assumed, in this segment, about 8% of patients are prescribed a JAK inhibitor at this point. With increasing evidence on efficacy and safety, we have modelled that the orally dosed targeted small molecules (such as JAK inhibitors) will take up an increasing part of the biologics market in the coming years. In our view, filgotinib has best-in-class potential and will become market leader with up to 35% market share in the JAK inhibitor space. This results in potential peak sales for filgotinib in RA of EUR 4bn in Europe and the US by 2026. Taking into account a tiered royalty rate in the range of 20-30% on US sales and co-promotion in the five major European countries and the Benelux as well as milestone payments, we arrive at a rNPV of EUR 2bn or EUR 39 per share. We risk-adjust FCFs to 75% at this stage in the development.

Exhibit 16 **Sensitivity analysis – assumptions for filgotinib in rheumatoid arthritis**

Market share	Probability of success				
	65.0%	70.0%	75%	85.0%	95.0%
15%	23.32	25.11	26.91	30.50	34.08
25%	28.42	30.61	32.79	37.17	41.54
35%	33.52	36.09	38.67	43.83	48.99
45%	38.61	41.58	44.55	50.49	56.43
55%	43.71	47.07	50.43	57.15	63.88

Price assumption	Probability of success				
	65.0%	70.0%	75%	85.0%	95.0%
-20%	26.49	28.52	30.56	34.64	38.71
-10%	29.82	32.11	34.41	39.00	43.58
0%	33.52	36.09	38.67	43.83	48.99
+10%	37.21	40.07	42.94	48.66	54.39
+20%	41.28	44.45	47.63	53.98	60.33

Source: Degroof Petercam estimates; As a base case we have included an annual price of EUR 12k in Europe and USD 20k in the US and 35% market share in the JAK inhibitor space.

- Inflammatory bowel disease: We have assumed about 35% of IBD patients will be treated with biologics, of which filgotinib could take a peak market share of 25%. These estimates imply peak sales of EUR 2.8bn for filgotinib in IBD. Including the terms of the agreement with Gilead as described for RA above, we arrive at a rNPV of EUR 657m (EUR 13/share) for Crohn's disease and EUR 482m (EUR 9/share) for ulcerative colitis. At this stage in the clinical development, we have again risk-adjusted FCFs to 75%.
- Additional indications: Galapagos and Gilead are targeting a broad label for filgotinib. We have included estimates for psoriatic arthritis and ankylosing spondylitis in our model, while we consider successes in the additional indications at this point as potential upside to our valuation. Following the positive Phase II readout of filgotinib in psoriatic arthritis, we increased the probability of success of this program to 30%. We estimate that this market could be a EUR 1.5bn opportunity in case a larger Phase III study confirms these initial results and the label expansion into psoriatic arthritis receives approval from the regulator. This leads to a rNPV of EUR 299m (EUR 6/share).

Building an IPF franchise

Devastating chronic progressive lung disease

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive lung disease that results in scarring (fibrosis) of the lungs. It is associated with increasing cough and shortness of breath and significantly impacts a patient's quality of life. The exact cause of the disease is unknown, a multitude of factors may contribute to its development. Risk factors include environmental elements, smoking, chronic viral infections, etc. There is also a strong genetic component involved and age is the dominant demographic risk factor. The disease typically affects adults over the age of 40. The prognosis for patients with IPF is poor; median survival is limited to 3-5 years.

Current treatments insufficient to alleviate unmet medical need

Management of IPF today primarily focusses on amelioration of symptoms, improving health status, preserving lung function, minimizing adverse effects of therapy and prolonging survival. Substantial progress in IPF management has been made in recent years following the update of the treatment guidelines in 2015. The international IPF guidelines were adapted with strong and conditional recommendations. Most importantly, combination treatment with prednisone (corticosteroid), azathioprine (immunosuppressant) and N-acetyl cysteine (mucolytic), used as standard of care, was strongly argued against owing to the association with harmful and adverse effects.

Another important step in IPF treatment was taken with the first successful randomized clinical trials that led in 2014 to the worldwide approval of two agents capable of impacting the natural course of IPF; pirfenidone (Esbriet; Roche) and nintedanib (Ofev; Boehringer Ingelheim). Both compounds have been shown to slow down the rate of functional decline in IPF, but neither pirfenidone nor nintedanib succeeded to demonstrate a significant survival benefit in IPF.

Exhibit 17 Phase III data that supported approval of Esbriet and Ofev

Trial	N° patients	Results
CAPACITY 004	435	Mean FVC change at week 72 was −8.0% in the pirfenidone group (2403 mg/day) and −12.4% in the placebo group
CAPACITY 006	344	Mean change in FVC at week 72 was −9.0% in the pirfenidone group and −9.6% in the placebo group (not significantly different)
ASCEND	555	The mean decline from baseline in FVC was 235 ml in the pirfenidone group and 428 ml in the placebo group at week 52
INPULSIS 1	515	The mean decline in FVC was 114.7 ml in nintedanib treated group and 239.9 ml in the placebo group at week 52
INPULSIS 2	513	The mean decline in FVC was 113.6 ml in nintedanib group and 207.3 ml in the placebo group at week 52

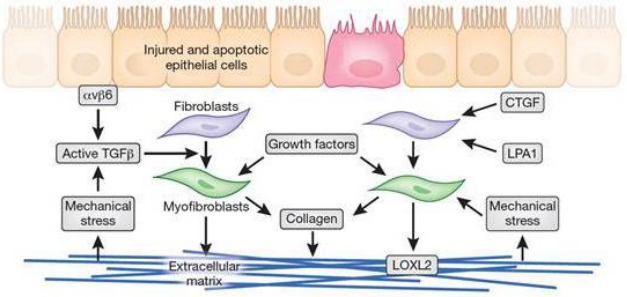
Source: Boehringer Ingelheim

Furthermore, these drugs often do not improve the symptoms of IPF patients and the disease continues to progress under treatment. Treatment with pirfenidone and nintedanib is associated with considerable adverse effects (e.g. severe diarrhoea and liver function test abnormalities with Ofev, nausea and rash with Esbriet). It is clear that there still is a large unmet medical need in IPF, which remains a major cause of morbidity and mortality. Nevertheless, Esbriet and Ofev reached up to USD 788m and USD 684m in 2016 sales, respectively.

Limited late-stage therapeutic candidates in development

Late-stage research into new therapeutics for the treatment of IPF remains limited. In August, Fibrogen disclosed positive results of its Phase II IPF trial with pamrevlumab, an antibody targeting connective tissue growth factor (CTGF). This factor is thought to be a critical mediator in the progression of fibrosis.

Exhibit 18 Processes driving IPF



Source: Garber K. *Nature Biotechnology* (2013)

Fibrogen's Phase II study enrolled 103 patients who were randomized (1:1) to receive either pamrevlumab or placebo for 48 weeks. Pamrevlumab met the primary efficacy endpoint of forced vital capacity percent predicted (ppFVC). The average decline in ppFVC from baseline to week 48 was 2.85 in the pamrevlumab arm, compared to 7.17 in the placebo arm. Pamrevlumab-treated patients had an average decrease in FVC of 129 ml at week 48, compared to 308 ml in patients receiving placebo.

Additionally, Fibrogen evaluated pamrevlumab in combination with approved IPF therapies. In these sub-studies, 36 patients on a stable dose of pirfenidone and 21 patients on nintedanib were randomized 2:1 to also receive pamrevlumab or placebo for 24 weeks. The combination was found to be well tolerated.

Exhibit 19 Overview of selected drug candidates

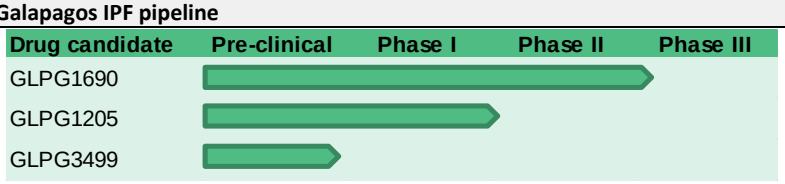
Company	Compound	Molecular mechanism	Stage	Results	Next milestone
Fibrogen	Pamrevlumab	Anti-CTGF mAb	Phase II	In a Phase II study with 103 patients the average decline in ppFVC from baseline to week 48 was 2.85, compared to 7.17 in the placebo arm	Initiation pivotal trial in 2018
Prometic Biosciences	PBI-4050	GPR40 and GPR84	Phase III	Mean change from baseline to week 12 in FVC in patients on PBI-4050 alone or PBI-4050 in combination with nintedanib was stable, -12 ml and +2 ml respectively	Phase III pivotal trial ongoing, with interim results expected at 26 weeks
Biogen	STX-100	anti-αvβ6 integrin receptor mAb	Phase II	Finished Phase II trial without reporting results	-
Promedior	PRM-151	rhPTX-2	Phase II	FVC and 6-min walk test showed trends towards improvement in the combined PRM-151 dose groups	Completion of placebo-controlled study expected in March 2019
Novartis / Morphosys	VAY736	anti-BAFF-R mAb	Phase II	-	Completion of placebo-controlled study expected in April 2022
Celgene	CC-90001	JNK inhibitor	Phase II	In a Phase Ib study with 16 patients, 83% showed increased FVC relative to baseline at 12 weeks	Phase II results of placebo-controlled study expected mid-2019
Bristol-Myers Squibb	BMS-986020	LPA1 antagonist	Phase II	-	Trial halted for unknown reasons
Roche	Lebrikizumab	anti-IL-13 mAb	Phase II	-	No progress noted since asset was sold to Dermira (August 2017)
Gilead	Simtuzumab	anti-LOXL-2 mAb	Phase II	-	Discontinued
Sanofi	SAR156597	Bispecific IL-4/IL-13 antibody	Phase II	-	Discontinued

Source: Company websites

Galapagos' IPF pipeline – three different modes of action

Galapagos' IPF portfolio contains three drug candidates, of which the most advanced is the autotaxin inhibitor GLPG1690. The target was identified by Galapagos using an inflammation assay in their proprietary target discovery platform. Autotaxin converts LPC to the bioactive lipid mediator LPA, which covers a family of related molecules (LPA18:2, LPA:4). In patients with IPF, increased autotaxin and LPA levels have been noted in bronchoalveolar lavage fluid. LPA also appears to be elevated in exhaled breath condensate.

Exhibit 20 **Galapagos IPF pipeline**

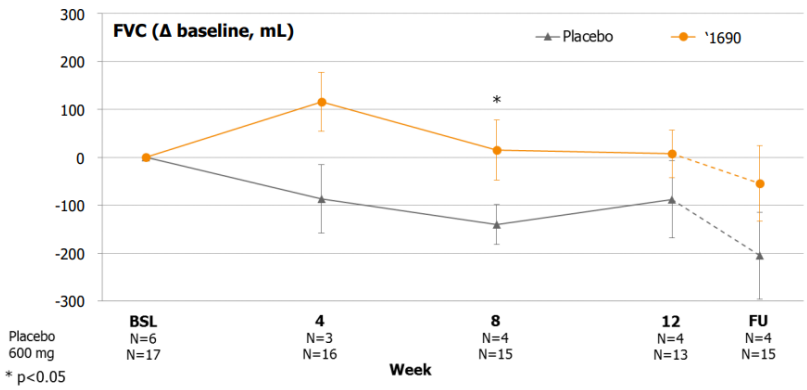


Source: Galapagos

In August 2017, Galapagos completed the Phase IIa (FLORA) study with its autotaxin inhibitor in IPF patients. The placebo-controlled study evaluated a once-daily oral dose of GLPG1690 (600 mg). Before screening, patients went through a wash out period of 4 weeks without pirfenidone or nintedanib. The compound was administered to 23 IPF patients, of which 17 received GLPG1690 and 6 placebo.

Over the 12-week treatment period, **patients in the GLPG1690-treated group showed stabilization of disease**, with and FVC increase of 8 ml compared to baseline, while patients in the placebo group showed an FVC reduction of 87 ml. Functional respiratory imaging (FRI) confirmed these results, showing statistically significant differences on two specific parameters. A steep reduction in plasma LPA18:2 was noted in the treatment group, pointing to GLPG1690 target engagement.

Exhibit 21 **FLORA Phase IIa results**



Source: Galapagos

GLPG1690 will now be evaluated in a pivotal Phase III program, which will consist of two identically designed trials, ISABELA 1 and ISABELA 2. The trials will enrol 1,500 patients in total diagnosed with IPF on top of their local standard of care, whether or not they were previously or currently treated with Esbriet (pirfenidone) and Ofev (nintedanib). The primary endpoint will be the rate of decline of FVC (in mL) until week 52. Secondary assessments will include respiratory-related hospitalizations, mortality, quality of life, safety and tolerability.

The design of the Phase III program, which will include treatment-naïve patients as well as patients on Ofev or Esbriet, should allow the company to obtain a **broad label**, potentially including monotherapy as well as add-on therapy. Taking into account at least one year of

recruitment and 12-24 months follow-up, we expect the trials to **read out around 2021**, making the path to market relatively short.

In addition to GLPG1690, the company is developing two additional product candidates for IPF, namely GLPG3499 and GLPG1205, both expected to enter clinical studies in 2018. The mechanism of action of the first compound has not been disclosed. The second is a GPR84 inhibitor discovered by Galapagos that was previously evaluated as product candidate for the treatment of ulcerative colitis. However, while showing a favourable tolerability profile, no effect of the compound could be detected in that indication. Interestingly, the safety profile has already been established in these earlier studies and the compound consequently immediately moved into Phase II for IPF. The PINTA study is a randomized, double-blind, placebo-controlled trial investigating a 100 mg once-daily oral dose. GLPG1205 or placebo will be administered for 26 weeks in up to 60 IPF patients. Patients may remain on their local standard of care as background therapy. Primary objective of the trial is to assess the change from baseline in Forced Vital Capacity (FVC in mL) over 26 weeks compared to placebo.

Short path to market

An estimated 200,000 patients are affected by IPF in Europe and the US. Despite limited efficacy and considerable adverse effects, Ofev and Esbriet are being adopted as standard treatment and combined sales reached USD 1.9bn in 2017. Both are priced at nearly USD 100k per patient per year.

Although the data on GLPG1690 are still limited and do not allow straightforward extrapolation to the Phase III trial, the demonstration of stable disease up to 12 weeks is encouraging for the evolution of GLPG1690 in IPF. We have assigned a probability of success of 40% to the program at this stage. As the compound will immediately move into a Phase III registration program, the path to market is relatively short; we expect GLPG1690 to reach the market by 2022. Taking into account a peak penetration of 20% and an annual price tag of USD 85k in the US and EUR 45k in Europe, we estimate peak sales could reach EUR 1.9bn by 2026. Our rNPV points to EUR 537m (EUR 10/share).

The cystic fibrosis triple combination program

Galapagos has been running an extensive program in cystic fibrosis (CF) in collaboration with AbbVie. The goal is to develop a therapy consisting of three components, the so called triple combination therapy.

CF can be attributed to different genetic mutations in the cystic fibrosis transmembrane conductance regulator (CFTR), involved in the production of sweat, digestive fluids and mucus. About 5% of CF patients have a 'gating mutation', most frequently this concerns a mutation called G155D. CF patients in this class generally benefit very well from Vertex Pharmaceuticals' potentiator, Kalydeco (ivacaftor). However, nearly 90% of CF patients have a F508del mutation, where Kalydeco is not effective. Based on the different mechanisms involved, a combination of ivacaftor and lumacaftor (Orkambi) was proposed. Despite the proven benefit of Orkambi in F508del homozygous patients (with two copies of the F508del mutation), there are still significant improvements needed to increase the efficacy and safety profile. Importantly, F508del heterozygous patients currently have no disease-modifying treatment option. Since February 2018, a third product (Symdeko; tezacaftor/ivacaftor and ivacaftor) entered the market after receiving FDA approval. Symdeko is indicated for CF patients aged 12 and older who have two copies of the F508del mutation in the CFTR gene or who have at least one mutation that is responsive to tezacaftor/ivacaftor.

Nevertheless, an efficacious therapy to address 90% of CF patients would need to consist of a combination of a potentiator and multiple correctors that work synergistically to correct the CFTR defect. In this field, Galapagos and Vertex are the main players.

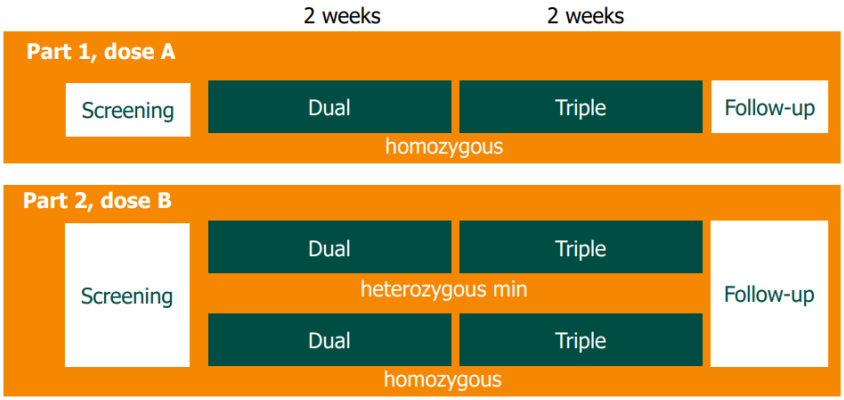
Galapagos initiated its first triple combination trial in April 2018. The FALCON study will evaluate '2451 (potentiator) +'2222 (C1 corrector) +'2737 (C2 corrector) in up to 24 CF patients. The Phase II open label trial is being conducted in multiple centres, initially in the UK with potential expansion to other European countries. **Topline results (part one of trial; low dose) are expected in 3Q18.**

The study will comprise two parts:

- Part one will entail treatment of eight patients for two weeks with a fixed dose dual combination '2451+'2222 in homozygous F508del patients. This will be followed by two weeks' treatment with '2451, '2222, and '2737.
- Part two will entail treatment for two weeks with a higher dose dual combination of '2451 and '2222 in separate eight-patient cohorts of homozygous F508del patients and heterozygous F508del patients with a minimal function mutation on the other allele. This will be followed by two weeks' treatment with '2451, '2222, and '2737.

Efficacy will be measured by changes in sweat chloride and percent predicted forced expiratory volume during the first second (ppFEV1%).

Exhibit 22 **FALCON trial design**



Source: Galapagos

PELICAN disappointed

The PELICAN trial that evaluated C2 corrector '2737 in homozygous patients on top of Orkambi recently produced disappointing results. The study recruited 22 adult CF patients homozygous for the Class II F508del mutation. Patients remained on a stable dose of Orkambi while receiving treatment with '2737 over a period of four weeks, with up to three weeks follow-up. The primary endpoint of the trial is the safety and tolerability of '2737. Secondary endpoints include measurements of sweat chloride and ppFEV%.

The mean change from baseline in sweat chloride for the '2737 treatment arm on day 28 versus placebo was a significant decrease of 19.6 mmol/L (p=0.02). The mean absolute change from baseline in ppFEV1 for the '2737 treatment arm versus placebo through day 28 was 3.4% (p=0.08). These results were below our expectations. We were hoping to see an improvement in FEV1 of at least 5% compared to Orkambi alone.

Though '2737 has been designed to work in concert with Galapagos' compounds and not Orkambi, a more pronounced improvement should have been observed in the PELICAN trial. Exposures of '2737 were in line with expectations, although drug-drug interactions could be noted.

On the back of the PELICAN trial results, Galapagos announced that AbbVie would not be initiating the second triple combination. While AbbVie seems to be backing out, Galapagos is keen to continue its research in CF and is in the process of reviewing the future of its CF collaboration with AbbVie.

Vertex has set the bar high

Vertex has now clearly taken the lead in the race towards a triple combination therapy. The company announced results of the Phase I and Phase II studies of different triple combination regimens in 2017 (Phase II data summarized below).

Exhibit 23 Summary of Vertex' key efficacy data

Data at day 29	Homozygous			Heterozygous		
	Absolute ppFEV1 From baseline	**Sweat Chloride**	**CFQ-R**	**Absolute ppFEV1** From baseline	**Sweat Chloride**	**CFQ-R**
VX-659 *in combination with teza/iva*	+9.7	-42.2	+19.5	+11.6	-43.7	+19.8
VX-445 *in combination with teza/iva*	+11	-39.6	+20.7	+13.8	-39.1	+25.7

Source: Vertex Pharmaceuticals

In February 2018, Vertex initiated its first Phase III triple combination study and the company is now running two Phase III trials, evaluating VX-659 and VX-445 in combination with tezacaftor and ivacaftor.

Vertex is also evaluating potentiator VX-561 (once-daily formulation of ivacaftor) in dose-ranging studies to develop a once-daily triple combination regimen. The initiation of a triple combination with this potentiator was slightly delayed (to 1Q19 from mid-2018) due to the request of the FDA for additional monotherapy/dose-ranging data following Phase II. The FDA seems to be adopting a stringent regulatory process, which could lead to longer development times than initially anticipated for Galapagos.

Room for two?

While in vitro data pointed to potential strong effects of Galapagos' compounds, it will be clearly difficult to surpass the high bar set by Vertex. Furthermore, the efficacy results of the C2 corrector '2737 in the PELICAN trial were disappointing, decreasing the chances of success of the triple combination that contains this component.

We previously adjusted our estimates for Galapagos' CF program to our bear case scenario taking into account the strong performance of Vertex. Following the PELICAN results, we decreased the probability of success for this program further to 10%, cutting the value of in our model from EUR 6 per share to EUR 2.

Technology platform keeps on delivering

In addition to the programs described above, Galapagos made strong progress with its product candidates for the treatment of atopic dermatitis (MOR106) and osteoarthritis (GLPG1972) as well with its earlier stage programs. The company continues to generate novel compounds through its discovery platform.

Targeting IL-17C in atopic dermatitis

Atopic dermatitis (AtD; eczema) is a common skin disorder, affecting approximately 20% of children (45% of patients are diagnosed by six months of age) and 1-3% of adults in industrialized countries. Historically, treatment of AtD consists of emollients in combination with topical corticosteroids and systemic therapy in patients with severe disease. However, topical therapies are frequently not adequate to treat and control symptoms in patients with moderate and severe disease and systemic immunosuppressive agents are often either not approved or not recommended.

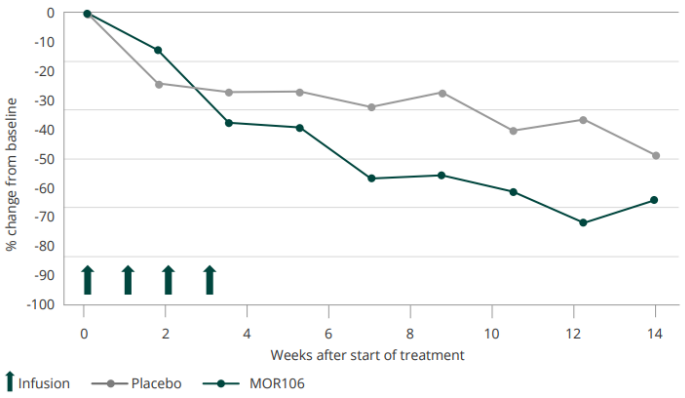
The treatment landscape in AtD is expected to change substantially in the near future. A number of biologics have been evaluated for their use in AtD, of which dupilumab (IL-4 Rα targeting mAb; Regeneron/Sanofi) was the first to obtain approval in 2017 for the treatment of adults with moderate-to-severe AtD whose disease is not adequately controlled with topical prescription therapies. Agents in clinical development include among others tralokinumab (anti-IL-13 mAb), ANB-020 (anti-IL-33 mAb), baricitinib (JAK inhibitor), BMS-981164 (anti-IL-31 mAb), etc.

Phase II trial to validate IL-17C mechanism of action

MOR106 is the first human monoclonal antibody against IL-17C in clinical development and was designed by MorphoSys on their Ylanthia platform. The target represents the third novel mode of action discovered by Galapagos.

Galapagos and AtD partner MorphoSys reported initial results of their placebo-controlled Phase Ib trial with MOR106 in September 2017. While still involving a small number of patients, the Phase Ib results gave a first indication of efficacy, demonstrating **83% (5 out of 6) patients reaching EASI-50 in the highest dose level** (vs. 17% or 1 out of 6 in the placebo group). This response seemed to be maintained after discontinuation of treatment (>2 months).

Exhibit 24 MOR106 Phase Ib EASI score (% change from baseline)



Source: Galapagos

The next phase in the clinical development of MOR106 was taken in May 2018, with the initiation of the Phase II IGUANA trial. A minimum of 180 patients with moderate-to-severe AtD will be treated with one of three different doses of MOR106 (1, 3 or 10 mg/kg) or placebo

using two different dosing regimens. Dosing at 2 or 4-week intervals will be evaluated over the 12-week treatment period, followed by a 16-week observation period. The primary endpoint of the trial is the percentage change from baseline in EASI score at week 12.

Atopic dermatitis market opportunity

The AtD market is estimated at about USD 3.7bn[4], but the use of topical corticosteroids in other skin indications as well as off-label use of systemic therapies for AtD may cloud the actual market value.

The worldwide atopic dermatitis population is estimated at a staggering 35 million patients, of which about 18% have moderate to severe disease. We have estimated that in this large and fragmented market, MOR106 could reach a 2% peak penetration, leading to EUR 1.7bn sales by 2028 (assuming pricing at par with dupilumab).

Global license agreement with Novartis

Galapagos and Morphosys entered into a worldwide, exclusive agreement with Novartis for their joint MOR106 program. The companies received an upfront payment of EUR 95m and potential milestone payments of up to approximately EUR 850m in addition to tiered royalties up to low-teens to low-twenties. Galapagos and Morphosys will share all payments equally. Novartis will bear all future research, development, manufacturing and commercialization costs related to MOR106.

Importantly, the companies see additional potential in indications outside atopic dermatitis. As such, they will cooperate to broaden the existing development plan for MOR106. Today, the program represents EUR 3 of our EUR 110 TP, but evidence of the benefit of MOR106 in additional indications holds significant further upside potential.

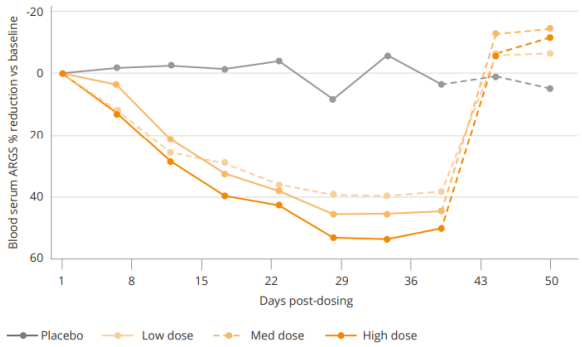
Positive biomarker data give way to global Phase IIb program in OA

Osteoarthritis (OA) is the most common joint disorder, with an estimated global prevalence of 8.2%[5]. Pharmacological treatments are mainly related to relief of symptoms and there is no disease-modifying OA drug (that is, treatment that will reduce symptoms in addition to slowing or stopping the disease progression) yet approved by the regulatory agencies.

Galapagos is developing GLPG1972 in collaboration with Servier. The compound acts on ADAMTS-5, an aggrecanase involved in the breakdown of aggrecan in joint cartilage. In a placebo-controlled, double-blind Phase Ib study including 30 OA patients, GLPG1972 was shown to be well tolerated. One treatment discontinuation was reported with reversible abnormal liver function test on day 15 in the highest dose cohort. Patients were treated during 4 weeks and further evaluated in a 3-week follow-up period. A secondary objective was the measurement of ARGS neoepitope blood levels, which is a key marker of cartilage breakdown. Patients on treatment showed a **dose-dependent reduction of ARGS neoepitope** vs. placebo. ARGS levels decreased by up to 53% below baseline in the 300 mg group.

[4] IMS Health
[5] Research and Markets - Global Osteoarthritis Market Spotlight 2017-2027

Exhibit 25 GLPG1972 Phase Ib results



Source: Galapagos

A global Phase II clinical program (ROCELLA) with GLPG1972 has been initiated in collaboration with Servier. ROCCELLA will be a multiregional, randomized, double-blind, placebo-controlled, dose ranging trial evaluating the efficacy and safety at week 52 of three different once-daily doses of S201086/GLPG1972 in patients with OA. ROCCELLA is planned to recruit approximately 850 patients in up to 15 countries. The trial will include measurement of cartilage thickness using quantitative magnetic resonance imaging. Secondary objectives include safety and tolerability, several additional measures of structural progression, changes in bone area, pain, function, stiffness, and patient global assessment. Galapagos will be responsible for ROCCELLA in the United States, where 300 patients are targeted to be recruited. Servier will run the trial in all other countries.

Market opportunity in OA

The vast majority of OA patients can be successfully treated with general pain medication. We estimated that about 8% of the large OA population will need more specific agents. Here, we assume GLPG1972 could capture up to 5% of market share, leading to peak sales around EUR 1.6bn by 2027. Galapagos will retain full rights in the US, while the company will receive royalties from Servier outside the US.

Based solely on biomarker data from a limited number of patients, we have attributed a probability of success of 25% to the OA program at this stage. This results in a rNPV of EUR 184m (EUR 3.6/share).

Profit & Loss (EUR m)	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
Revenues	**90.0**	**60.6**	**151.6**	**155.9**	**183.8**	**151.3**	**146.2**
(of which Sales)	0.0	0.0	0.0	0.0	0.0	0.0	31.2
(of which Other revenues)	90.0	60.6	151.6	155.9	183.8	151.3	115.0
Gross profit	**90.0**	**60.6**	**151.6**	**155.9**	**183.8**	**151.3**	**144.7**
Operating costs	-126.6	-150.0	-163.1	-245.7	-317.4	-349.0	-368.0
(of which R & D)	-111.1	-129.7	-139.6	-218.5	-284.1	-312.5	-328.1
EBIT	**-36.6**	**-89.4**	**-11.5**	**-89.8**	**-133.6**	**-197.6**	**-223.3**
Net Financial Result	1.4	-30.2	65.7	-25.7	-5.0	-7.0	-8.7
Pre-tax result	**-35.2**	**-119.6**	**54.2**	**-115.5**	**-138.7**	**-204.7**	**-232.1**
Taxes	-2.1	1.2	-0.2	-0.2	-0.2	-0.2	-0.2
Except. / Discont. operations	67.5	-	-	-	-	-	-
Associates	-	-	-	-	-	-	-
Minorities	-	-	-	-	-	-	-
Net declared earnings	**30.2**	**-118.4**	**54.0**	**-115.7**	**-138.9**	**-204.9**	**-232.3**

Cash Flow (EUR m)	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
EBIT	-36.6	-89.4	-11.5	-89.8	-133.6	-197.6	-223.3
Depreciation	3.6	2.4	3.3	3.6	3.8	3.9	4.1
Amortization	0.0	1.0	0.9	0.7	0.5	0.4	0.3
Impairment	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Changes in provision	0.0	-0.1	0.0	0.0	0.0	0.0	0.0
Changes in working capital	-54.5	-34.0	235.0	-78.6	-134.6	-27.3	-40.0
Others	3.2	4.6	12.5	16.2	16.9	17.2	18.6
Operational Cash Flow	**-84.4**	**-115.5**	**240.1**	**-147.9**	**-247.0**	**-203.4**	**-240.3**
Tax expenses	0.0	0.0	0.0	-0.2	0.0	0.0	0.0
Dividends from associates	-	-	-	-	-	-	-
Net interest charges	8.8	1.0	-0.7	1.1	-5.0	-7.0	-8.7
Others	-	-	-	-	-	-	-
CF from operating activities	**-75.6**	**-114.6**	**239.4**	**-147.0**	**-252.1**	**-210.4**	**-249.1**
CAPEX	-2.1	-6.1	-4.5	-5.3	-7.4	-6.1	-5.8
Investments in intangibles	-0.7	-0.5	-0.3	-2.1	-0.5	-0.4	-0.4
Acquisitions	130.8	0.0	0.0	0.0	0.0	0.0	0.0
Divestments	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Others	-7.4	2.3	0.2	0.0	0.0	0.0	0.0
CF from investing activities	**120.6**	**-4.3**	**-4.5**	**-7.4**	**-7.9**	**-6.5**	**-6.3**
Dividend payment	-	-	-	-	-	-	-
Minor. & pref. dividends	-	-	-	-	-	-	-
Equity financing	4.4	271.4	396.0	353.4	0.0	0.0	0.0
Others	0.0	0.0	0.0	-0.1	0.0	0.0	0.0
CF from financing activities	**4.4**	**271.4**	**396.0**	**353.4**	**0.0**	**0.0**	**0.0**
Changes in consolidation scope	-	-	-	-	-	-	-
Exchange rate impact	0.0	0.0	4.8	-27.8	0.0	0.0	0.0
Net debt/cash change	**-49.5**	**-152.5**	**-630.9**	**-198.9**	**259.9**	**216.9**	**255.4**

Notes Company reports and Degroof Petercam estimates

Balance Sheet (EUR m)	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
Fixed assets	**12.4**	**68.0**	**76.1**	**88.6**	**95.6**	**101.2**	**106.9**
Tangible fixed assets	10.1	13.8	15.0	16.7	20.3	22.4	24.2
Goodwill	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other intang. assets	2.0	1.6	1.0	2.5	2.5	2.5	2.6
Financial fixed assets	0.3	52.7	60.1	69.4	72.8	76.4	80.1
Other fixed assets	-	-	-	-	-	-	-
Current assets	**208.7**	**360.6**	**1,000.0**	**1,191.2**	**920.3**	**704.4**	**450.1**
Inventories	0.3	0.3	0.3	0.3	0.0	0.0	0.0
Trade receivables	10.6	13.1	19.9	39.7	28.0	28.0	28.0
Other current assets	-	-	-	-	-	-	-
Cash & Equivalents	197.8	347.2	979.8	1,151.2	892.4	676.5	422.1
Discontinued assets	-	-	-	-	-	-	-
Total assets	**270.2**	**442.5**	**1,083.3**	**1,286.3**	**1,112.3**	**862.5**	**595.3**
Total Equity	**206.1**	**365.0**	**758.7**	**1,012.0**	**873.1**	**668.3**	**436.0**
Equity	206.1	365.0	758.7	1,012.0	873.1	668.3	436.0
Minorities & preferred	-	-	-	-	-	-	-
Provisions	2.9	2.7	3.6	3.6	3.7	3.7	3.7
Provisions for pensions	2.9	2.7	3.5	3.6	3.6	3.6	3.6
Deferred taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other provisions	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Other LT liabilities	0.9	2.3	2.5	1.6	0.0	0.0	0.0
LT interest bearing debt	0.9	2.3	2.5	1.6	0.0	0.0	0.0
Current liabilities	**32.7**	**30.0**	**103.8**	**171.7**	**165.5**	**160.5**	**155.5**
ST interest bearing debt	0.1	0.1	0.1	0.0	0.0	0.0	0.0
Accounts payables	32.6	30.0	103.7	171.7	165.5	160.5	155.5
Other ST liabilities	0.1	0.0	0.0	0.0	0.0	0.0	0.0
Discontinued liabilities	-	-	-	-	-	-	-
Total liabilities	**270.2**	**442.5**	**1,083.3**	**1,286.3**	**1,112.3**	**862.5**	**595.3**

EV and CE details (EUR m)	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
Market cap.	**459.5**	**2,218.0**	**2,818.8**	**4,019.7**	**4,504.6**	**4,504.6**	**4,504.6**
+ Net financial debt	-197.7	-347.1	-979.8	-1,151.2	-892.4	-676.5	-422.1
(of which LT debt)	0.9	2.3	2.5	1.6	0.0	0.0	0.0
(of which ST debt)	0.1	0.1	0.1	0.0	0.0	0.0	0.0
(of which Cash position)	197.8	347.2	979.8	1,151.2	892.4	676.5	422.1
+ Provisions (pension)	-	-	-	-	-	-	-
+ Minorities (MV)	-	-	-	-	-	-	-
- Peripheral assets (MV)	-	-	-	-	-	-	-
+ Others	-	-	-	-	-	-	-
Enterprise Value	**261.8**	**1,870.9**	**1,839.1**	**2,868.5**	**3,612.2**	**3,828.1**	**4,082.5**
Equity (group share)	**206.1**	**365.0**	**758.7**	**1,012.0**	**873.1**	**668.3**	**436.0**
+ Net financial debt	-197.7	-347.1	-979.8	-1,151.2	-892.4	-676.5	-422.1
+ Provisions (pension)	2.9	2.7	3.5	3.6	3.6	3.6	3.6
+ Minorities	-	-	-	-	-	-	-
- Peripheral assets	-	-	-	-	-	-	-
+ Others	-	-	-	-	-	-	-
Capital employed (for ROCE)	**11.3**	**20.6**	**-217.5**	**-135.6**	**-15.6**	**-4.6**	**17.5**
+ Accumulated goodwill amortiz.	-	-	-	-	-	-	-
CE (for ROCE grossed gdwll)	**11.3**	**20.6**	**-217.5**	**-135.6**	**-15.6**	**-4.6**	**17.5**

Notes Company reports and Degroof Petercam estimates

Per Common Share (EUR)	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
Declared EPS	**1.02**	**-3.03**	**1.17**	**-2.27**	**-2.71**	**-4.00**	**-4.53**
Declared EPS (fully diluted)	1.02	-3.03	1.17	-2.27	-2.71	-4.00	-4.53
CFS	-	-	-	-	-	-	-
Dividend	-	-	-	-	-	-	-
Book Value	6.95	9.34	16.40	19.88	17.04	13.04	8.51
Shares (m)							
At the end of F.Y.	29.665	39.076	46.256	50.896	51.235	51.235	51.235
Average number	29.665	39.076	46.256	50.896	51.235	51.235	51.235
Fully diluted Average number	29.665	39.076	46.256	50.896	51.235	51.235	51.235

Ratios	12/14	12/15	12/16	12/17	12/18e	12/19e	12/20e
P/E	15.2	nm	52.2	nm	nm	nm	nm
P/CF	-	-	-	-	-	-	-
P/BV	2.2	6.1	3.7	4.0	5.2	6.7	10.3
EV/Revenues	2.9	30.9	12.1	18.4	19.7	25.3	27.9
EV/R & D	2.4	14.4	13.2	13.1	12.7	12.3	12.4
EV/EBIT	-7.1	-20.9	-160.0	-31.9	-27.0	-19.4	-18.3
EV/CE	23.1	90.7	-8.5	-21.1	-230.9	-833.4	232.9
Dividend yield	-	-	-	-	-	-	-

Notes Company reports and Degroof Petercam estimates

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	SELL	REDUCE	HOLD	ADD	BUY
High Beta >= 1.3	RP<-15%	-15%<=RP<-6%	-6%<=RP<+6%	+6%<=RP<+15%	RP>=15%
Medium 0.9 < Beta > 1.3	RP<-10%	-10%<=RP<-4%	-4%<=RP<+4%	+4%<=RP<+10%	RP>=10%
Low Beta <= 0.9	RP<-6%	-6%<=RP<-2%	-2%<=RP<+2%	+2%<=RP<+6%	RP>=6%

RP : Relative Performance against Degroof Petercam coverage universe

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