Filgotinib in RA - we see an €2.5bn opportunity

- **Addressable population in RA**: In 2018, we assume there are c. 2.1m patients in the US diagnosed with RA, c. 1.9m in the EU big 5, c. 0.2m in BeNeLux and c. 0.7m in Japan, of which 70% are treated and 80% have moderate to high disease activity. Of these moderate to severe patients, we assume 40% use of bDMARDs in the US and Japan, compared to 15% in the EU big 5 and BeNeLux, with the remainder of the patients using csDMARDs. Furthermore, we assume an annual new diagnosis rate of 40/100,000 across all regions.

- **Market setting**:
  1st line – MTX naïve: We assume all newly diagnosed patients in the 1st line are solely treated with csDMARDs, as we believe prescribers are used to using cheap MTX as a first line treatment, and expect this trend to continue.
  2nd line – csDMARD intolerant or inadequate responder: We assume that c. 60% of patients in the 2nd line show intolerance or inadequate response to csDMARDs in the US, compared to 35% ex-US (due to a higher willingness to switch / greater availability of bDMARDs), of which the majority are treated by anti-TNF and fewer treated with IL-6(R) inhibitors and JAK inhibitors.
  3rd line – bDMARDs intolerant or inadequate responder: We assume that 35% of patients in the 3rd line show intolerance or inadequate response to bDMARDs, of which the majority are treated using Xeljanz.

- **Market penetration**:
  1st line – MTX naïve: We have assumed that filgotinib will capture 0% of the 1st line (MTX naïve) market across all regions.
  2nd line – csDMARD intolerant or inadequate responder: In the US, we see filgotinib capturing 1% starting 2020 and 12.5% from 2025 and beyond. Across the EU big 5 and BeNeLux, we see Filgotinib capturing 1% starting 2021 and 12.5% from 2026 and beyond.
  3rd line – bDMARDs intolerant or inadequate responder: In the US, we see Filgotinib capturing 2.5% starting 2020 and 15% from 2023 and beyond. Across the EU big 5 and BeNeLux, we see Filgotinib capturing 2.5% starting 2021 and 20% from 2027 and beyond. In Japan, we see Filgotinib capturing 2% of patients starting 2021 and 15% from 2026 and beyond.

- **Filgotinib pricing**: Given the benefit demonstrated by filgotinib, we assume it will be priced in line with Xeljanz at $27,905 in 2018 (assuming 30% rebates and 80% compliance adjustment) and keep the price flat going forward. In EU and BeNeLux we assume a 50% discount to the US price and in RoW (Japan) we assume a 20% discount. With filgotinib's composition of matter patent expiring in March, 2034, we assume 50% loss of sales across all regions, with biologics entering starting 2Q'34.

- **Peak revenues**: Based on the above assumptions, we forecast unadjusted global peak sales estimates in RA of €2.5bn; €2.3bn risk adjusted.

- **Valuation**: Despite the strong DARWIN 1 and 2 Phase II data and read across from other assets in the JAK inhibitors class, we apply a 90% probability of success to sales as we believe a small amount of clinical trial risk remains. Filgotinib sales, royalties and milestone in RA are collectively worth €60.6 per share / 51% of our EmV.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Table 18: Summary RA model for 2020-2026E

Summary Model for RA - US	2020E	2021E	2022E	2023E	2024E	2025E	2026E
- Use of csDMARDs	60%	60%	60%	60%	60%	60%	60%
Total Patients on csDMARDs	729,193	734,297	739,437	744,613	749,826	755,075	760,360
1st line - MTX naïve							
Newly diagnosed patients	133,552	134,487	135,428	136,376	137,331	138,292	139,260
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
2nd line - csDMARDs intolerant or inadequate responder							
2nd line patients on csDMARDs	595,641	599,811	604,009	608,237	612,495	616,782	621,100
- csDMARDs failure rate	54%	54%	54%	54%	54%	54%	54%
Patients who fail on csDMARDs	321,549	323,800	326,066	328,349	330,647	332,962	335,293
- Filgotinib penetration	0.3%	1.5%	3.0%	6.0%	9.0%	12.5%	12.5%
Filgotinib patients	804	4,857	9,782	19,701	29,758	41,620	41,912
Revenue ($m)	22	136	273	550	830	1,161	1,170
Revenue (€m)	19	117	235	473	714	999	1,006
3rd line - bDMARDs intolerant or inadequate responder							
- Use of bDMARDs	40%	40%	40%	40%	40%	40%	40%
Patients on bDMARDs	486,129	489,532	492,958	496,409	499,884	503,383	506,907
- bDMARDs failure rate	35%	35%	35%	35%	35%	35%	35%
Patients who fail on bDMARDs	170,145	171,336	172,535	173,743	174,959	176,184	177,417
- Filgotinib penetration	1.0%	5.0%	8.0%	10.0%	12.5%	15.0%	15.0%
Filgotinib patients	1,701	8,567	13,803	17,374	21,870	26,428	26,613
Revenue ($m)	47	239	385	485	610	737	743
Revenue (€m)	40	206	331	417	525	634	639
Total US Revenue ($m)	**70**	**375**	**658**	**1,035**	**1,441**	**1,899**	**1,912**
Total US Revenue (€m)	**59**	**322**	**566**	**890**	**1,239**	**1,633**	**1,644**
Summary Model for RA - EU5 & BeNeLux	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**
- Use of csDMARDs	85%	85%	85%	85%	85%	85%	85%
Total Patients on csDMARDs	1,033,023	1,040,254	1,047,536	1,054,869	1,062,253	1,069,689	1,077,177
1st line - MTX naïve							
Newly diagnosed patients	140,325	140,699	141,074	141,452	141,831	142,212	142,595
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
2nd line - csDMARDs intolerant or inadequate responder							
2nd line patients on csDMARDs	892,698	899,556	906,462	913,417	920,422	927,477	934,581
- csDMARDs failure rate	35%	35%	35%	35%	35%	35%	35%
Patients who fail on csDMARDs	312,444	314,845	317,262	319,696	322,148	324,617	327,103
- Filgotinib penetration	0.0%	1.0%	2.5%	5.0%	7.5%	10.0%	12.5%
Filgotinib patients	0	3,148	7,932	15,985	24,161	32,462	40,888
Revenue ($m)	0	44	111	223	337	453	570
Revenue (€m)	0	38	95	192	290	390	491
3rd line - bDMARDs intolerant or inadequate responder							
- Use of bDMARDs	15%	15%	15%	15%	15%	15%	15%
Patients on bDMARDs	161,974	162,380	162,787	163,196	163,608	164,021	164,437
- bDMARDs failure rate	35%	35%	35%	35%	35%	35%	35%
Patients who fail on bDMARDs	56,691	56,833	56,975	57,119	57,263	57,407	57,553
- Filgotinib penetration	0.0%	2.5%	7.5%	5.0%	10.0%	15.0%	17.5%
Filgotinib patients	0	1,421	4,273	2,856	5,726	8,611	10,072
Revenue ($m)	0	20	60	40	80	120	141
Revenue (€m)	0	17	51	34	69	103	121
Total EU5 & BeNeLux Revenue ($m)	**0**	**64**	**170**	**263**	**417**	**573**	**711**
Total EU5 & BeNeLux Revenue (€m)	**0**	**55**	**146**	**226**	**359**	**493**	**611**
Summary Model for RA - Japan	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**
- Use of csDMARDs	60%	60%	60%	60%	60%	60%	60%
Total Patients on csDMARDs	225,857	225,857	225,857	225,857	225,857	225,857	225,857
1st line - MTX naïve							
Newly diagnosed patients	50,800	50,800	50,800	50,800	50,800	50,800	50,800
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
2nd line - csDMARDs intolerant or inadequate responder							

2nd line patients on csDMARDs	175,057	175,057	175,057	175,057	175,057	175,057	175,057
- csDMARDs failure rate	35%	35%	35%	35%	35%	35%	35%
Patients who fail on csDMARDs	61,270	61,270	61,270	61,270	61,270	61,270	61,270
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
3rd line - bDMARDs intolerant or inadequate responder							
- Use of bDMARDs	40%	40%	40%	40%	40%	40%	40%
Patients on bDMARDs	150,571	150,571	150,571	150,571	150,571	150,571	150,571
- bDMARDs failure rate	35%	35%	35%	35%	35%	35%	35%
Patients who fail on bDMARDs	52,700	52,700	52,700	52,700	52,700	52,700	52,700
- Filgotinib penetration	0.0%	2.0%	5.0%	7.5%	10.0%	12.0%	15.0%
Filgotinib patients	0	1,054	2,635	3,952	5,270	6,324	7,905
Revenue ($m)	0	24	59	88	118	141	176
Revenue (€m)	0	20	51	76	101	121	152
Total Japan Revenue ($m)	**0**	**24**	**59**	**88**	**118**	**141**	**176**
Total Japan Revenue (€m)	**0**	**20**	**51**	**76**	**101**	**121**	**152**
Global RA Revenue ($m)	**70**	**462**	**887**	**1,386**	**1,975**	**2,613**	**2,800**
Global RA Revenue (€m)	**59**	**397**	**763**	**1,192**	**1,699**	**2,247**	**2,408**

Source: J.P. Morgan estimates

Drug: Filgotinib

Mechanism: JAK-1 inhibitor

Partner: Gilead

Next catalyst: Data from Phase III DIVERSITY-1 and SELECTION-1 in 2020

Peak Sales: €0.5bn in CD (€0.3bn risk adjusted) and €0.7bn in UC (€0.3bn risk adjusted)

Risk adjustment: 60% in UC and 40% in UC

EmV: €7.3 for CD (6%) and €7.7 for UC (6%)

Royalty: 20-30% tiered, 50% profit share (EU5 and BeNeLux)

Inflammatory Bowel Disease – €1.2bn opportunity

Filgotinib is also being developed for the treatment of Inflammatory Bowel Disease (IBD) in the DIVERSITY Phase III study for Crohn Disease (CD) and the SELECTION Phase III study for Ulcerative Colitis (UC). Filgotinib has completed the Phase II FITZROY trial in CD as monotherapy, demonstrating potential best in class efficacy and, although we have yet to see any data in UC, DMC recommended no changes to the study protocol following a planned futility analysis. Key competitors include approved anti-TNFs (e.g. AbbVie's Humira) and other JAK inhibitors in clinical development, primarily AbbVie's upadacitinib in Phase III trials for both CD and UC. We model €0.5bn in peak CD sales, which we include at a 60% probability of success (€0.3bn), and model €0.7bn in peak UC sales, which we include at a 40% probability of success (€0.3bn)

Phase III development programme

DIVERSITY and SELECTION will test 100mg and 200mg/qd filgotinib versus placebo in moderate to severe CD and UC, respectively, involve c. 1,300 patients each (Table 19). In May, 2018, GLPG and GILD announced the progression of SELECTION into Phase III following recommendation by the Data Monitoring Committee (DMC) based on the a planned interim futility analysis conducted after 350 patients completion the induction period in the Phase IIb portion of the study. Management further noted that the male subjects in the US will only be eligible to receive the 200mg dose if they have failed ≥ anti-TNF and Entyvio (vedolizumab), but insisted that this dose restriction was not driven by any toxicity signals seen in the Phase II study of filgotinib in CD (FITZROY).

Table 19: Phase III programs in IBD

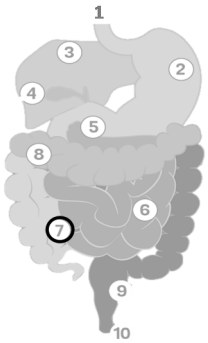
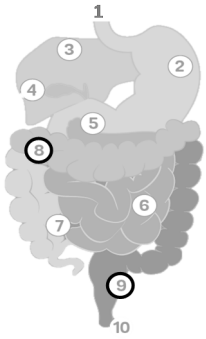
Molecule	Program	Indication	Patient Population	N	Arms	Duration	Primary endpoint	Secondary endpoints	Time line
NCT02914561	DIVERSITY-1	CD	Mod-Sev active UC, biologic-naïve and experienced	1,320	1) Dose A (100mg/qd) 2) Dose B (200mg/qd) 2) Placebo	58 weeks	1) Clinical remission by PRO2 at week 10 and 58 2) Endoscopic response at week 10 and 58	1) Clinical remission by CDAI at week 10 and 58 2) PRO2 and endoscopic response at week 10 3) PK	2019
NCT02914522	SELECTION-1	UC	Mod-Sev active UC, biologic-naïve and experienced	1,300	1) Dose A (100mg/qd) 2) Dose B (200mg/qd) 2) Placebo	58 weeks	1) Remission based on components of MCS at week 10 and 58	1) MCS remission at week 10 2) Histologic remission at week 10	2019

CD = Crohn's Disease; UC = Ulcerative Colitis; PRO2 = Patient Reported Outcomes; MCS = Mayo Clinic Score
Source: Company press releases, Clinicaltrials.gov

IBD is caused by inflammation along the digestive tract

IBD includes CD and UC, both of which cause chronic inflammation in the gastrointestinal (GI) tract and share similar symptoms, including abdominal pain, lose stools (diarrhea), fatigue, weight loss and anemia (a reduced level of red blood cells) due to rectal bleeding. Although GI tract inflammation is common across CD and UC, there are distinct differences between the two diseases:

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Disease	Crohn's Disease	Ulcerative Colitis
Sites of inflammation	• Can affect any part of the GI tract but most commonly affects the end of the small intestine • Inflammation can extend through entire thickness of the gastrointestinal wall	• limited to the large intestine and rectum • Inflammation only occurs in innermost layer of gastrointestinal wall
1) Oesophagus 2) Stomach 3) Liver 4) Gall Bladder 5) Pancreas 6) Small intestine 7) End of ileum (last part of small intestine) 8) Colon (large intestine) 9) Rectum 10) Anus		
Population/ incidence	780,000 Americans* 1 in every 650 in the UK**	907,000 Americans* 1 in every 420 people in the UK**
Onset	• Can occur at any age, but usually appears for the first time between the ages of 10 and 40 • Slightly more common in women	• Can occur at any age, but usually appears for the first time between the ages of 15 and 25 • Affects women and men equally
Measure of disease activity	• Severity of CD is often measured using the Crohn's Disease Activity Index (CDAI) ○ 150-220 = mild-moderate disease ○ 220-450 = moderate-severe disease ○ >450 = severe/fulminant disease ○ <150 = clinical remission	• Severity of UC is often measured using the Mayo Clinic Score (MCS) (12-point scale) ○ 3-5 = mild disease ○ 6-10 = moderate disease ○ 11-12 = severe disease ○ ≤2 and no subscore >1 = clinical remission

*According to the Crohn's and Colitis Foundation of America; ** According to Crohn's and Colitis UK

Current treatment paradigm highlights readiness to adopt JAK inhibitors

At present there is no cure for CD or UC but, once diagnosed, the symptoms can often be effectively managed with drugs (see Table 20) and sometimes surgery; however, CD and UC are chronic illnesses, and changes are likely to occur over time. There are currently five main categories of medications used to treat IBD:

1) **Aminosalicylates** (5-ASAs): Given orally or rectally to decrease inflammation of the lining of the intestine and primarily used to treat UC. Examples include mesalazine, olsalazine, sulphalazine and balsalazide.

2) **Steroids**: Keep the immune system in check by blocking the substances that trigger inflammatory responses. Effective for short-term control of flare-ups, but not recommended for maintenance treatment due to possible side-effects. Examples include prednisone, prednisolone, and budesonide.

3) **csDMARDs**: Suppress the immune system to reduce inflammation and used to maintain remission in patients who have not responded to 5-ASAs and steroids or only responded to steroids.

4) **Antibiotics**: Metronidazole and ciprofloxacin are sometimes used in CD when infections occur or after surgery, but there is no substantial scientific evidence to support the use of antibiotics in the treatment of UC.

5) **bDMARDs**: Indicated for patients with moderate to severe disease who have not responded well to conventional therapy. Anti-TNF drugs prevent inflammation by targeting TNF, while Tysabri and Entyvio stop white blood cells from entering the lining of the gut and causing inflammation and Stelara targets two proteins in the body (IL-12 and IL-23) which cause inflammation. We note that Tysabri is

only approved in patients with elevated levels of C-reactive protein (CRP) and is rarely used given concerns around risk of progressive multifocal leukoencephalopathy (PML; an opportunistic viral infection of the brain) as seen in multiple sclerosis patients.

6) **tsDMARD** (JAK-inhibitors).

Patients who do not respond well to medication will be advised to consider surgery. According to the Crohn's & Colitis Foundation of America (CCFA), after 30 years of disease, up to a third of patients with UC will require surgery while about 70% of patients with CD eventually require surgery. The most common type of surgery for UC patients is a colectomy and ileostomy, which removes the colon and brings the lower end of the small intestine out through an opening in the wall of the abdomen, known as a stoma. This compares to CD patients, where the two most common types of operations are stricturoplasty and resection. Stricturoplasty operations can be used to widen narrowed parts of the intestine, while resection operations involve removes the severally inflamed parts of the intestine and joins the healthy ends back together.

Table 20: Summary of Inflammatory Bowel Disease biologic therapies

Product (Molecule)	Indication	Company	FDA Approval	Target	2017 Global sales* ($m)
bDMARDs					
Anti-TNF					
Remicade (infliximab)	CD & UC	J&J	1998 / 2005	TNF	6,315
Humira (adalimumab)	CD & UC	AbbVie	2007 / 2012	TNF	18,427
Cimzia (certolizumab)	CD	UCB	2008	TNF	1,609
Simponi (golimumab)	UC	J&J	2013	TNF	1,833
Non-TNF					
Tysabri (natalizumab)	CD	Biogen	2008	α4-integrin	1,973
Entyvio (vedolizumab)	CD & UC	Takeda	2014	α4β7-integrin	1,796
Stelara (ustekinumab)	CD	J&J	2016	IL-12/23	4,011
Anti-TNF biosimilars					
INFLECTRA (incliximab-dyyb)	CD & UC	Pfizer	2016	TNF	
AMJEVITA (adalimumab-atto)	CD & UC	Amgen	2016	TNF	
RENFLEXIS (infliximab-abda)	CD & UC	Merck & Co	2017	TNF	
CYLTEZO (adalimumab-adbm)	CD & UC	Boehringer Ingelheim	2017	TNF	
IXIFI (infliximab-qbtx)	CD & UC	Pfizer	2017	TNF	
tsDMARDs					
Xeljanz (tofacitinib)	UC	Pfizer	2018	JAK-1/3	
Upadacitinib	CD & UC	AbbVie	Expected 2020 / 2021	JAK-1/2/3	

Source: Company Data. *includes sales from non-IBD indications; converted to USD to 2017 weighted avg. FX where applicable

Phase II/III data highlight favorable activity in anti-TNF naive patients and comparable activity in anti-TNF experienced patients vs. competition

In December, 2015, GLPG announced interim 10 week results from the Phase II study of filgotinib in patients with moderate to severe CD (FITZROY) (see Table 21).This study tested 175 patients, either anti-TNF naive or anti-TNF failures, receiving 200mg/qd (n = 128) or placebo (n = 44) for 20 weeks. The study met its primary endpoint of clinical remission at week 10, with 48% filgotinib-treated patients reporting clinical remission versus 23% in the placebo group (p = 0.0067). Clinical response, defined as a CDAI decrease ≥100 points, was also statistically significant; 60% in filgotinib-treated patients versus 41% for placebo (p = 0.0045). Overall, efficacy data stacks up well against competitor trials (see Table 21). Furthermore, we note that the mean baseline CDAIs of 291 and 299 in the drug and placebo arm, respectively, are comparable to the ranges seen for competitor trials.

Looking at the split of the data for anti-TNF naive patients (n = 73) and anti-TNF failures (n = 101), it becomes evident that the overall benefit is driven by the data from the TNF naive population and that the high placebo reported in anti-TNF failures may skew the results. More specifically, anti-TNF naive patients treated with filgotinib achieved a placebo-adjusted clinical remission of 48% versus 8% in anti-TNF failures. We acknowledge that anti-TNF failures represent a more challenging patient population; however, the clinical response of 29% reported in the placebo group is at the upper end of competitor trials ranging from 7% to 36%.

On safety profile, filgotinib was reported as being well tolerated, with similar incidences in SAEs and AEs were observed between the two arms and he majority of SAEs related to worsening of CD. Filgotinib was reported to be well tolerated, with similar rates of AEs across the two arms and no cases of opportunistic infections, tuberculosis, EVT or malignancies reported. That being said, there was one case of serious infection (pneumonia), which resulted in death, and one case of herpes zoster in the filgotinib group. Management also noted that tested male hormones extensively and saw no changes in the hormone levels of males receiving the 200mg/qd dose level.

Table 21: Phase II Filgotinib efficacy data in CD vs. competitor data

Molecule	Filgotinib		Upadacitinib			Tofacitinib			Adalimumab			
Brand name	-		-			Xeljanz			Humira			
Target	JAK-1		JAK-1			JAK-1/3			TNF			
Source	10W FITZROY		16W CELEST			8W Panes et al., 2017			4W GAIN		4W CLASSIC-1	
Trial No.	NCT02048618		NCT02365649			NCT01393626			NCT00105300			
Phase	II		II			IIb			III			
Population	58% anti-TNF experienced; 42% anti-TNF naïve		96% anti-TNF experienced			77% anti-TNF experienced			previously treated with Infliximab		anti-TNF naïve	
Baseline CDAI	291	299	303			314	320	313	313	313		
n	128	44	36	35	37	86	86	91	159	166	76	74
Dose	200 mg/qd	Pbo	24mg/bid	24mg/qd	Pbo	5mg/bid	10mg/bid	Pbo	Total SQ	Pbo	160/80mg wk0/2 SQ	Pbo
Efficacy data (n, %)												
Clinical remission*	48%	23%	31%	20%	16%	44%	43%	37%	21%	7%	36%	12%
Placebo-adjusted	25%		15%	4%		7%	6%		14%		24%	
p-value	*0.0067*								*<0.001*		*0.001*	
Clinical response**	60%	41%	56%	31%	27%	71%	69%	54%	38%	25%	50%	25%
Placebo-adjusted	19%		29%	4%		16%	14%		13%		25%	
p-value	*0.0386*		*<0.05*			*<0.05*					0.002	

Clinical remission defined as CDAI < 150; Clinical response defined as a 100-point reduction in CDAI
Source: Company data

The use of JAK inhibitors in CD was questioned when Pfizer announced underwhelming data for a placebo-controlled Phase II study of Xeljanz (tofacitinib) in CD, the first JAK inhibitor to be moved to clinic in this indication. The induction study tested 261 subjects with moderate to severe CD treated with Xeljanz (tofacitinib) dosed at 5mg/bid (n = 85) or 10 mg/bid (n = 86) or placebo (n =90) twice daily for 8 weeks. More specifically, neither of the two Xeljanz (tofacitinib) doses tested showing a significant impact on clinical remission, which constituted the primary endpoint of the induction study, with placebo responder rate (37%) being comparable to rates seen for the 5mg/bid dose (44%) and the 10mg/bid dose (43%). However, we believe these results are specific to Xeljanz (tofacitinib) and not a reflection of JAK inhibitors as a whole: In fact we believe filgotinib could have a differentiated profile in this indication, with anemia being one of the most common complications of IBD and filgotinib being the only JAK inhibitor to have shown a dose-dependent increase in Hb levels in RA patients

Awaiting data in UC

We have not seen any data yet for filgotinib in UC; however, in May, 2018, GLPG received a $15m payment from GILD for progression of the SELECTION study in UC into Phase III. This was recommended by the Data Monitoring Committee (DMC) based on a planned interim futility analysis conducted after 350 patients completed the induction period in the Phase 2b portion of the study. We note that we are always awaiting data for ABBV's upadacitinib, currently in Phase IIb.

Filgotinib in IBD - we see an €1.2bn opportunity

- **Addressable population in IBD**: According to the Crohns Colitis Foundation, there are currently 780k and 907k Americans with CD and UC, respectively. Across EU big 5 and Benelux, we assume there are currently c. 1.1m patients with CD and c. 1.1m and UC. Of these patients, we assume 56% have moderate to high disease activity, 25% of which are in long term remission across all regions. Of the people not in clinical remission, we assume 24% of are treated with biologics (1L therapy) in the US compared to 17% across EU big 5 and Benelux, with the remainder of the patients on non-biologics (2L therapy).

- **Market setting & penetration**:
  1st line – biologics: In CD, we have assumed that filgotinib will capture 2% of patients starting 2023 and 10% from 2027 and beyond in the US and 1% starting 2024 and 7% from 2028 and beyond across EU big 5 and BeNeLux. In UC, we have taken a conservative view and assumed that anti-TNF will remain standard-of-care (SoC) in 1L therapy, with filgotinib capturing 0% of the 1st line market across all regions.
  2nd line – non-biologics: In CD, we see filgotinib capturing 0% of the 2nd line market across all regions. In UC, we have assumed that filgotinib will capture 2% of the patients starting 2023 and 8% from 2026 and beyond in the US, offset by a year across EU big 5 and BeNeLux (2% in 2024; 8% in 2027 and beyond).

- **Filgotinib pricing**: Given the benefit demonstrated by filgotinib, we assume it will be priced in line with tofacitinib (Xeljanz®) at $27,905 in 2018 (assuming 30% rebates and 80% compliance adjustment) and keep the price flat going forward. In EU big 5 and Benelux we assume a 40% discount to the US price and in RoW (Japan) we have applied a 20% discount to the US price. With filgotinib's composition of matter patent expiring in March, 2023, we have cut the price by 50% in 2034 across all regions.

- **Peak revenues**: Based on the above assumptions, we forecast unadjusted global filgotinib peak sales estimates of $0.6bn (€0.5bn) in CD and $0.8bn (€0.7bn) in UC. On a risk adjusted basis, we forecast peak sales of $0.4bn (€0.3bn) and $0.3bn ($0.3bn) in CD and UC, respectively.

- **Valuation**: Despite the strong FITZROY Phase II data, we apply a 60% probability of success to CD sales given the historical failure of tofacitinib (Xeljanz®) in this indication and remaining clinical trial risk. Similarly, despite progression into Phase III based on the completed interim futility analysis with 350 ulcerative colitis patients, we apply a 40% probability of success to UC sales given the lack of clinical data available to date. Filgotinib sales and royalties in CD are worth €7.3 per share / 6% of our EmV, comparable to sales and royalties in UC worth €7.7 per share / 6% of our EmV. Collectively, IBD is worth €15 per share / 13% of our EmV.

Table 22: Summary CD model for 2020-2026E

Summary Model for CD - US	2020E	2021E	2022E	2023E	2024E	2025E	2026E
1st line - patients on biologic							
Patients on biologic/1L therapy	144,851	152,722	158,402	159,531	160,668	161,814	162,967
- Filgotinib penetration	0.0%	0.0%	1.5%	3.0%	5.0%	7.5%	10.0%
Filgotinib patients	0	0	2,376	4,786	8,033	12,136	16,297
Revenue ($m)	0	0	66	134	224	339	455
Revenue (€m)	0	0	57	115	193	291	391
2nd line - patients on non-biologic							
Patients on non-biologic/2L therapy	194,643	189,193	185,950	187,276	188,611	189,955	191,310
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
Total US Revenue ($m)	**0**	**0**	**66**	**134**	**224**	**339**	**455**
Total US Revenue (€m)	**0**	**0**	**57**	**115**	**193**	**291**	**391**
Summary Model for CD - EU5 & BeNeLux	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**
1st line - patients on biologic							
Patients on biologic/1L therapy	111,979	115,232	115,540	115,849	116,160	116,472	116,786
- Filgotinib penetration	0.0%	0.0%	0.0%	1.0%	2.0%	4.0%	6.0%
Filgotinib patients	0	0	0	1,158	2,323	4,659	7,007
Revenue ($m)	0	0	0	19	39	78	117
Revenue (€m)	0	0	0	17	33	67	101
2nd line - patients on non-biologic							
Patients on non-biologic/2L therapy	271,108	268,875	269,593	270,314	271,039	271,768	272,500
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
Total EU5 & BeNeLux Revenue ($m)	**0**	**0**	**0**	**19**	**39**	**78**	**117**
Total EU5 & BeNeLux Revenue (€m)	**0**	**0**	**0**	**17**	**33**	**67**	**101**
Global CD Revenue ($m)	**0**	**0**	**66**	**153**	**263**	**417**	**572**
Global CD Revenue (€m)	**0**	**0**	**57**	**132**	**226**	**358**	**492**

Source: J.P. Morgan estimates

Table 23: Summary UC model for 2020-26E

Summary Model for UC - US	2020E	2021E	2022E	2023E	2024E	2025E	2026E
1st line - patients on biologic							
Patients on biologic/1L therapy	168,435	177,588	184,193	185,506	186,829	188,161	189,502
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
2nd line - patients on non-biologic							
Patients on non-biologic/2L therapy	226,335	219,997	216,227	217,768	219,321	220,884	222,459
- Filgotinib penetration	0.0%	0.0%	1.5%	3.0%	5.0%	7.5%	7.5%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	91	182	306	462	466
Revenue (€m)	0	0	78	157	263	398	400
Total US Revenue ($m)	**0**	**0**	**91**	**182**	**306**	**462**	**466**
Total US Revenue (€m)	**0**	**0**	**78**	**157**	**263**	**398**	**400**
Summary Model for UC - EU5 & BeNeLux	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**
1st line - patients on biologic							
Patients on biologic/1L therapy	111,979	115,232	115,540	115,849	116,160	116,472	116,786
- Filgotinib penetration	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Filgotinib patients	0	0	0	0	0	0	0
Revenue ($m)	0	0	0	0	0	0	0
Revenue (€m)	0	0	0	0	0	0	0
2nd line - patients on non-biologic							
Patients on non-biologic/2L therapy	271,108	268,875	269,593	270,314	271,039	271,768	272,500
- Filgotinib penetration	0.0%	0.0%	0.0%	1.5%	3.0%	5.0%	7.5%
Filgotinib patients	0	0	0	4,397	8,131	13,588	20,437
Revenue ($m)	0	0	0	74	136	228	342
Revenue (€m)	0	0	0	63	117	196	294
Total US Revenue ($m)	**0**	**0**	**0**	**74**	**136**	**228**	**342**
Total US Revenue (€m)	**0**	**0**	**0**	**63**	**117**	**196**	**294**
Global UC Revenue ($m)	**0**	**0**	**91**	**256**	**442**	**690**	**808**
Global UC Revenue (€m)	**0**	**0**	**78**	**220**	**380**	**593**	**695**
Global IBD Revenue ($m)	**0**	**0**	**157**	**409**	**705**	**1,106**	**1,380**
Global IBD Revenue (€m)	**0**	**0**	**135**	**352**	**606**	**952**	**1,187**

Source: J.P. Morgan estimates

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Drug: Filgotinib

Mechanism: JAK-1 inhibitor

Partner: Gilead

Next catalyst: Data from Phase III DIVERSITY-1 and SELECTION-1 in 2020

Peak Sales: €0.3bn in PsA (€0.2bn risk adjusted) and €0.3bn in AS (€0.2bn risk adjusted)

Risk adjustment: 50% in PsA and 20% in AS

EmV: €3.5 for PsA (3%) and €3.8 for AS (3%)

Royalty: 20-30% tiered, 50% profit share (EU5 and BeNeLux)

Pipeline in a Product – Further autoimmune indications could boost the peak sales potential of filgotinib

Psoriatic Arthritis – €0.3bn opportunity

Psoriatic Arthritis (PsA) is a type of arthritis that develops in some people who suffer from psoriasis and causes inflammation in the joints. According to the World Psoriasis Day consortium, 125m (2-3%) suffer from psoriasis worldwide, 10-30% of which develop PsA according to the National Psoriasis Foundation. PsA usually develops between the ages of 30-50, but can develop at any age, and symptoms include red and scaly skin (psoriasis), stiff joints and swollen fingers or toes (arthritis).

In September, 2018, GLPG and GILD announced results from the Phase II study of filgotinib in adults with moderate to severe PsA (EQUATOR). The study tested 131 patients receiving 200mg/qd (n = 62) or placebo (n = 62) for 16 weeks. Although early, the efficacy stacks up favorably to the competition (see Table 24). Specifically, the study met its primary endpoint of ACR20 at week 16, with filgotinib-treated patients reporting an ACR20 response of 80% versus 33% for placebo ($p<0.001$). ACR50 and ACR70 responses were also statistically significant; 48% versus 15% for placebo ($p<0.001$) and 23% versus 6% for placebo ($p<0.01$), respectively. Filgotinib was reported to be well tolerated, with similar rates of adverse events across the two arms and no cases of opportunistic infections, tuberculosis, EVT or malignancies reported. That being said, there was one case of serious infection (pneumonia), which resulted in death, and one case of herpes zoster in the filgotinib group.

Table 24: Phase II EQUATOR 16W data vs. competitor data in PsA

Molecule	Filgotinib		Xeljanz						Cosentyx			Tremfya		Humira	
Target	JAK-1		JAK-1/3			JAK-1/3			IL-17			IL-23		TNF	
Patient population	mod-severe active PsA (85% TNF naïve)		mod-severe PsA (TNF-naïve)			mod-severe PsA (TNF-IR)			mod-severe PsA			active PsA		mod-severe PsA	
Phase	II	II	III	III	III	III	III	III	III	III	III	IIb	IIb	III	III
N	65	65	107	104	105	131	132	131	100	100	98	100	49	151	162
Dose		Pbo	5mg /q2d	10mg /q2d	Pbo	5mg /q2d	10mg /q2d	Pbo	150mg Wk 0, 1, 2, 3 & 4 - then q4w	300mg Wk 0, 1, 2, 3 & 4 - then q4w	Pbo	100mg Wk 0 & 4 - then q8w	Pbo	40mg /q2w	Pbo
Efficacy	**Week 16**		**Month 3**			**Month 3**			**Week 16**			**Week 24**		**Week 12**	
ACR20	80%	33%	50%	61%	33%	50%	47%	24%	60%	57%	18%	58%	18%	58%	14%
Pbo-adj.	47%		17%	28%		26%	23%		42%	39%		40%		44%	
p-value	*<0.001*														
ACR50	48%	15%	28%	40%	10%	30%	28%	15%	37%	35%	6%			36%	4%
Pbo-adj.	**33%**		**18%**	**30%**		**15%**	**13%**		**31%**	**29%**				**32%**	
p-value	*<0.001*														
ACR70	23%	6%	17%	14%	5%	17%	14%	10%	17%	15%	2%			20%	1%
Pbo-adj.	17%		12%	9%		7%	4%		15%	13%				19%	
p-value	*<0.01*														

Source: Company data

Overall, we view these data as encouraging, and potentially differentiated, but await detailed results, to be presented at a future scientific conference, and competitor readouts (AbbVie's upadacitinib currently in Phase III) to better understand the competitive landscape and positioning for filgotinib. While many cases of PsA are well treated with biologics (e.g. Humira) the size of the markets allows for the entry of new differentiated therapies. We currently forecast peak sales of €0.3bn ($0.4bn)

in PsA and apply a 50% probability of success. Filgotinib sales in PsA are worth €3.5 per share / 3% of our EmV.

Ankylosing Spondylitis – €0.3bn opportunity

Ankylosing Spondylitis (SA) is a form of inflammatory arthritis that primarily affects the spine, causing inflammation of the vertebrae and impairing spinal mobility. According to a paper published in Rheumatology in 2014 and based on 36 eligible studies, the mean AS prevalence per 10,000 was 23.8 in Europe, 16.7 in Asia, 31.9 in North America, 10.2 in Latin America and 7.4 in Africa (between 0.1% and 1.4% globally). Symptoms usually start to appear between the ages of 17 and 45, and include frequent lower back pain, mild fever, loss of appetite and general discomfort.

In September, 2018, GLPG and GILD announced results from the Phase II study of filgotinib in adults with moderate to severe active AS (TORTUGA). The study tested 116 patients receiving filgotinib at a dose of 200mg/qd (n = 58) or placebo (n = 58) for 12 weeks. The efficacy looks comparable to Pfizer's Xeljanz (tofacitinib) (see Table 25), with filgotinib-treated patients reporting a placebo-adjusted mean change from baseline (CFB) in AS Disease Activity Score (ASDAS) of -0.9 ($p<0.0001$), compared to -0.7 ($p\leq0.001$) reported for the Xeljanz (tofacitinib) dosed at 5mg/big and 10mg/bid. Filgotinib-treated patients also reported an ASAS20 response of 76% versus 40% for placebo ($p<0.0001$), comparable to patients treated with Xeljanz (tofacitinib) dosed at 5mg/qd showing 81% versus 41% for placebo ($p<0.001$). Filgotinib was reported to be well tolerated in AS, with the majority of adverse events categorized as mild to moderate and reported in equal proportion between the two arms. However, there was one serious adverse event (pneumonia) reported in the filgotinib group. Additionally, a non-serious DVT was reported in a patient who had an inherited risk of thrombosis and was treated with filgotinib.

Table 25: Phase II TORTUGA AS 12W data vs. competitor data in AS

Molecule Brand name Target Patient Population	Filgotinib - JAK-1 Mod-severe active AS		Tofacitinib Xeljanz JAK-1/3 Active AS				Secukinumab Cosentyx IL-17 Active AS despite DMARDs (33% TNF failures)		Adalimumab Humira TNF Mod-severe AS	
Phase	II		II				III	III	III	III
N	58	58	52	52	52	52	72	98	151	162
Dose	200mg/qd	Pbo	2mg/bid	5mg/bid	10mg/bid	Pbo	150mg Wk 0, 1, 2, 3 & 4 - then q4w	Pbo	40mg/q2w	Pbo
Efficacy	**Week 12**		**Week 12**				**Week 16**		**Week 12**	
ASDAS	-1.5	-0.6	-1.2	-1.4	-1.4	-0.7				
Placebo-adjusted	-0.9		-0.5	-0.7	-0.7					
p-value	*<0.0001*		*≤0.01*	*≤0.001*	*≤0.001*					
ASAS20	76%	40%	52%	81%	56%	41%	61%	28%	58%	21%
Placebo-adjusted	**36%**		**11%**	**40%**	**15%**		**33%**		**37%**	
p-value	*<0.0001*			*P<0.001*						
ASAS40			42%	46%	39%	20%	36%	11%	38%	10%
Placebo-adjusted			23%	27%	19%		25%		28%	
p-value										
ASAS70									23%	5%
Placebo-adjusted									18%	
p-value										

Source: Company data

We await detailed results, to be presented at a future scientific conference, and competitor readouts (AbbVie's upadacitinib currently in Phase III) to better understand the competitive landscape and positioning for filgotinib. We currently forecast peak sales of €0.3bn ($0.4bn) in AS and apply a 50% probability of success. Filgotinib sales in AS are worth €3.8 per share / 3% of our EmV

Additional inflammation indications for filgotinib could offer further upside.

Galapagos and Gilead have started Phase II trials in small bowel CD, Fistulizing CD, Sjogren's syndrome, Cutaneous Lupus, Systemic Lupus and Uveitis. Other JAK inhibitors have also started trials in Atopic Dermatitis, Axial Spondyloarthritis, Alopecia Areata and Psoriasis. We currently do not include any value for these additional indications in out model, and so sales in these areas could represent upside to our in market filgotinib peak sales of €4.4bn (€3.2bn risk adjusted).

Table 26: Additional filgotinib Phase II indications

Product	Indication	Target	Partner	Development Stage
Filgotinib	Small bowel CD	JAK 1	Gilead	Phase II
Filgotinib	Fistulizing CD	JAK 1	Gilead	Phase II
Filgotinib	Sjogren's	JAK 1	Gilead	Phase II
Filgotinib	Cutaneous lupus	JAK 1	Gilead	Phase II
Filgotinib	Lupus nephropathy	JAK 1	Gilead	Phase II
Filgotinib	Uveitis	JAK 1	Gilead	Phase II

Source: Company data

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

GLPG1690: Could be the first asset to halt disease progression in IPF

Drug: GLPG1690

Mechanism: Autotaxin inhibitor

Indication: Idiopathic Pulmonary Fibrosis

Next catalyst: Start of Phase III ISABELA program in 2H'18

Peak Sales: €1.0bn (€0.4bn risk adjusted)

Risk adjustment: 40%

EmV: €23.2 (19%)

GLPG currently have two fully proprietary small molecules in clinical development for IPF, each with distinct modes of action; namely, GLPG1690 (autotaxin inhibitor) and GLPG1205 (GPR84 inhibitor). Their most advanced molecule, GLPG1690, has received orphan drug designation in both the US and EU, and the Phase III ISABELA programme is expected to start in 2H'18.

GLPG1690 has completed the Phase IIa FLORA study as monotherapy in IPF patients, with the 12 week data presented at the American Thoracic Society (ATS) Conference in May, 2018. Based on the data available (Table 28), we believe GLPG1690 demonstrates a strong profile versus currently marketed drugs; namely, Roche's Esbriet (pirfenidone) and Boehringer Ingelheim's Ofev (nintedanib). The promising efficacy profile, coupled with once daily dosing, should position GLPG1690 well; however, we acknowledge that replication of data remains a key risk given the small number patients recruited in the Phase IIa FLORA study (n = 23). Hence, while we model potential peak sales of $1.1bn (€1.0bn) we use only a 40% probability of success.

IPF is an inflammatory lung disease with average survival of only 2-5 years from diagnosis

IPF is a chronic respiratory disease caused by the progressive build-up of scar tissue in the lungs and with a median survival of 2-5 years after diagnosis. This causes the lungs to become stiffer and reduces the efficiency of breathing, as measured by forced vital capacity (FVC); one of the markers of chronic disease progression and the preferred primary end point in IPF treatment trials. According to an article published in the European Respiratory Review in 2012, IPF affects approximately 3 million people worldwide, with the highest prevalence observed in patients ≥70 years of age, although IPF can affect people of all ages. Symptoms associated with IPF include getting out of breath when exerting oneself, having a persistent cough and feeling very fatigued.

There are currently two drugs approved for use in IPF; namely, Roche's Esbriet (pirfenidone) and Boehringer Ingelheim's Ofev (nintedanib), both of which have been shown to slow the development of scar tissue in the lungs and thus slow disease progression in IPF patients. However, with both drugs failing to stabilise FVC and being known for their significant side effect profiles, there remains a significant unmet medical need. While patients treated with Esbriet (pirfenidone) 2403mg/day across phase III trials had a higher incidence of photosensitivity reactions compared to those treated with placebo (9% vs. 1%), gastrointestinal disorders are highlighted on both labels. More specifically, the most common adverse reaction in patients treated with Ofev (nintedanib) across phase III trials was diarrhea (62% vs. 18% in placebo arm), leading to discontinuation in 5% of Ofev-treated patients.

Novel Mechanism of action could differentiate the GLPG IPF portfolio

GLPG1690 is an Autotaxin (ATX) inhibitor. ATX is the main enzyme responsible for the production of lysophosphatidic acid (LPA), which signals through various receptors to control a range of cell activities involved in inflammatory processes. LPA has specifically been linked to the pathophysiology of IPF, with increased LPA

and ATX levels having been found in the broncho alveolar lavage fluid and lung tissue of IPF patients, respectively, making ATX an attractive target for drug development

GPR84 (the target of GLPG1205) is a G-protein-coupled receptor further that binds median-chain free fatty acid ligands and is associated with inflammatory disorders. In May, 2018, a study published in the American Journal of Pathology demonstrated the relevance of GPR80 and GPR84 in fibrosis development. More specifically, the study showed that Prometic Life Sciences' PBI-4050 attenuated fibrosis in kidney, liver, heart, lung, pancreas and skin fibrosis models. PBI-4050 is a fatty acid analog that binds GPR40 and GPR84, activating GPR40 activity while suppressing GPR84 activity. Therefore, GPR40 and GPR84 may represent promising molecular targets in fibrosis pathways.

GLPG1690 appears to be the only drug to halt disease progression in Phase II clinical trials, although this needs to be confirmed in the ISABELA programme

On August 9th, 2017, GLPG announced positive topline results from the Phase IIa FLORA study of GLPG1690 as monotherapy in IPF patients. Initial FVC data for GLPG1690 demonstrated a strong profile versus currently marketed IPF drugs, with GLPG1690 halting disease progression compared to Esbriet (pirfenidone) and Ofev (nintedanib) which only show a drop in the speed of FVC decline across Phase III trials. More specifically, GLPG1690-treated patients (n = 17) experienced a 8mL increase in FVC at 12 weeks, while patients on placebo (n = 6) experienced a 87mL decrease from baseline (p = 0.3), which is in line with placebo response seen in other Phase III IPF studies (see Table 28). Although the small patient population (n = 23) limits inferences, a loose comparison of FVC improvement at c. 12 weeks suggests that GLPG1690 results in a c. 2x greater FVC change from baseline (CFB) versus Esbriet (pirfenidone) and Ofev (nintedanib). On the Phase IIa FLORA results webcast, management noted that the majority of GLPG-1690 treated patients reported a positive FVC and that they did not believe the differences in baseline disease characteristics between the active treatment and placebo groups (Table 27) impacted the efficacy outcomes.

Table 27: Baseline disease characteristics for the Phase IIa FLORA study

Baseline disease characteristics (mean)	Placebo	GLPG1690	Total
N	6	17	23
Duration of IPF (yrs)	1	1.9	1.7
DLCO (% predicted of normal)	40.6	37.8	38.6
Baseline FVC (L)	2.693	2.777	2.755
Baseline FVC (% predicted of normal)	69.7	75.3	73.8

Source: Company data

Although this study was designed to evaluate the safety and tolerability as opposed to the efficacy of GLPG1690, disease stabilisation was further supported by functional respiratory imaging (FRI) results, with GLPG1690-treated patients achieving statistical significance on two parameters; specific airway volume (p = 0.0137) and specific airways resistance (p = 0.0255) at total lung capacity. More specifically, mean CFB in specific airway volume was 0.079mL/L in GLPG1960-treated patients (n = 15) versus 3.038mL/L in the placebo arm (n = 3), and mean CFB in specific airway resistance was 0.004kPa/s in GLPG1960-treated patients (n = 14) versus -0.035kPa/s in the placebo arm (n = 3). Increased airway volume and decreased airway resistance were taken to be signs of reduced disease progression.

In addition to Esbriet and Ofev, data has been presented for Fibrogen's pamrevlumab, an antibody targeting connective tissue growth factor (CTGF), which Fibrogen believe could be a central mediator of tissue remodeling and fibrosis. The Phase II PRAISE trial pamrevlumab met the primary endpoint of percentage reduction in predicted FVC with an average decline of 2.85% in the pamrevlumab group, which was statistically smaller than the average decline of 7.17% in the placebo arm. So while the pamrevlumab data is encouraging it still does not halt the disease progression.

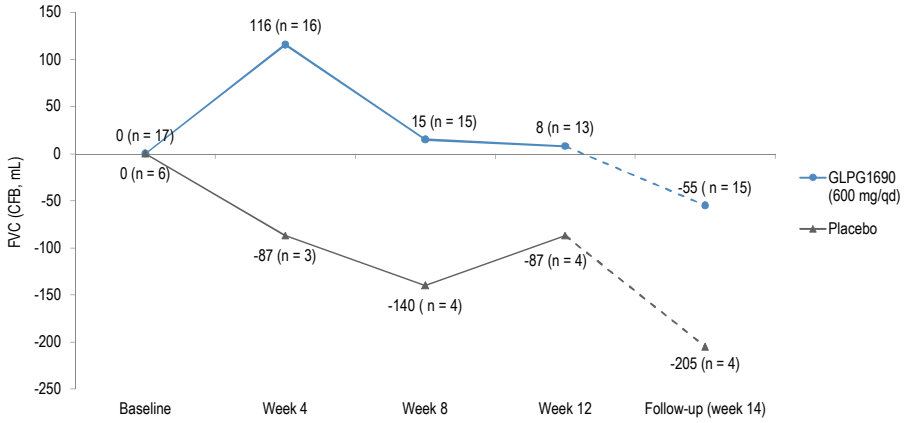
Table 28: GLPG1690 Phase IIa FLORA trial data vs. Esbriet and Ofev Phase III data

Molecule MoA Brand name Source Trial No.	GLPG'1690 Autotaxin inhibitor - 12W FLORA NCT02738801		Pirfenidone* Pyridone Esbriet 52W ASCEND*** NCT01366209		Nintedanib** Tyrosine kinase inhibitor Ofev 52W INPULSIS-1**** NCT01335464	
Phase	IIa		III		III	
N	17	6	278	277	309	204
Dose	600 mg/qd	Pbo	2403 mg/qd	Pbo	150 mg/bid	Pbo
FVC (mean CFB, mL)						
Week 4	116	-87			3	-8
Pbo adj.	203				10	
p-value	*0.13*					
Week 8	15	-140				
Pbo adj.	155					
p-value	*0.009*					
Week 12	8	-87	-38	-100	-18	-70
Pbo adj.	95		62		53	
p-value	*0.3*					
Follow-up (week 14 – GLPG1690 discontinued)	-55	-205				
Pbo adj.	150					
p-value	*0.06*					
Week 52			-235	-428	-95	-205
Pbo adj.			193		110	
Safety data (n, %)						
AEs	65%	67%	97%	91%	94%	87%
SAEs	6%	33%	20%	25%	31%	27%
AEs leading to discontinuation	6%	17%	15%	10%	21%	15%
AEs leading to death	0%	0%	4%	7%	-	-
Gastrointestinal AEs of special interest						
Dyspepsia	12%	17%	19%	7%	-	-
Diarrhea	6%	33%	26%	20%	62%	18%
Vomiting	-	-	13%	6%	12%	3%
Nausea	-	-	36%	16%	24%	7%
Abdominal pain	-	-	24%	15%	15%	6%

* AEs leading to discontinuation and death and AEs of special interest from pooled Phase III trials (ASCEND, CAPACITY-1 and -2); Esbriet 2403 mg/qd (n = 623), placebo (n = 624)
** AEs leading to discontinuation and death and AEs of special interest form pooled Phase III trials (TOMORROW, INPULSIS-1 and -2); Ofev 150 mg/bid (n = 723), placebo (n = 508)
*** Week 13 data estimated from chart
**** Week 4 and 12 data estimated from chart
Source: Company data

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Figure 17: FVC stabilization with GLPG1690 vs. placebo



Source: Company Data

While this initial data looks very encouraging, we note that it should be interpreted with some caution given the small number of patients recruited (n = 23), particularly in the placebo group (n = 6). Furthermore, changes in the number of patients for the FVC measurements at week 4, 8 12 and 14 makes the data more difficult to interpret (Figure 17). Note, GLPG 1690 was discontinued as per study protocol at week 12 leading to the decrease in the follow up period - however the benefit vs. placebo still remains.

GLPG1690 appears well tolerated, which is another advantage over standard therapies

In terms of safety profile, GLPG1690 appeared to be well-tolerated by IPF patients, with no significant differences reported between the treatment and placebo arms (see Table 28). Importantly, GLPG-1690 treated patients showed lower instances of gastrointestinal AEs, which are included under warnings in the Esbriet (pirfenidone) and Ofev (nintedanib) labels. On the Phase IIa FLORA webcast, management further noted that did not see any lab abnormalities or exacerbations over the course of the study.

Phase III development program announced for GLPG1690

The impressive results from the FLORA trial have led to GLPG progressing GLPG1690 to Phase III development and GLPG announced the design of their first independent Phase III program to evaluate GLPG1690 in IPF patients (ISABELA). This program will consist of two identical trials; ISABELA-1 and ISABELA-2, each of which will enroll 750 patients and include two doses of GLPG1690 vs. placebo (see Table 29). We expect the trials to start in 2H'18, and intend for it to support both New Drug Application (NDA) and Market Authorization Application (MAA) submission for a broad label, potentially including mono- and combination therapy. GLPG have not disclosed both GLPG1690 doses to be tested, but stated in their 1Q'18 earnings call that the top dose will be the one used in the Phase II FLORA study (600 mg/qd) and the other dose will be a lower dose.

In July 2018, GLPG announced the design of the Phase II PINTA trial of GLPG1205 in IPF patients (see Table 29). Contrary to GLPG1690, which is a selective autotaxin (AUX) inhibitor, GLPG1205 works via a different mechanism as a selective GPR84 inhibitor.

Table 29: GLPG Phase II/III programs in IPF

Molecule	Target	Program	Ph	Patient Population	N	Arms	Duration	Primary endpoint	Secondary endpoint	Trial Start
GLPG1690	Autotaxin	ISABELA-1	III	• IPF patients on SoC as background therapy • Global study with substantial US and EU components	750	1) Dose A (600mg/qd) 2) Dose B 3) Placebo	Patients will continue treatment until last patient in their respective study has completed 52 weeks of treatment	FVC (in mL) at week 52	1) Hospitalizations 2) Mortality 3) Quality of life 4) Safety/tolerability	3Q'18
GLPG1690	Autotaxin	ISABELA-2	III	• IPF patients on SoC as background therapy • Global study with substantial US and EU components	750	1) Dose A (600mg/qd) 2) Dose B 3) Placebo	Patients will continue treatment until last patient in their respective study has completed 52 weeks of treatment	FVC (in mL) at week 52	1) Hospitalizations 2) Mortality 3) Quality of life 4) Safety/tolerability	3Q'18
GLPG1205	GPR84	PINTA	II	• IPF patients on SoC as background therapy • Recruitment planned in 10 countries in Europe, North America and the Middle East	60	1) 100 mg/qd 2) Placebo	26 weeks	FVC (in mL) at week 26	1) Safety/tolerability 2) Pharmacokinetics & pharmacodynamics 3) Time to major events 4) Δ FEC 5) Quality of life	2H'18

SoC = Standard of Care i.e. Roche Esbriet® (pirfenidone) or Boehringer Ingelheim's Ofev® (nintedanib); FVC =Forced Vital Capacity; FEC = Functional Exercise Capacity
Source: Company press releases

To date, the largest Phase III program to have been completed in IPF is Roche / Genentech's Phase III program for Esbriet (pirfenidone), which involved 3 Phase III trials (ASCEND, CAPACITY-1 and -2) across >1,300 IPF patients. For this program, the recruitment time for the individual Phase III trials ranged from c. 12 months to c. 21 months (according to ClinicalTrials.gov). Although, GLPG's Phase III ISABELA program is larger, we note two key advantages to the protocol design:

- **Patients will remain on Standard of Care (SoC) throughout the trial**: The global Phase III program will enroll IPF patients on top of their local SoC, whether or not they were previously or currently are treated with Esbriet (pirfenidone) or Ofev (nintedanib), which we believe should help speed enrollment.
- **Potential to poach patients without access to SoC or waiting for reimbursement:** Management have stated that the recruitment for the Phase III program will be worldwide, with a significant proportion of patients in the US and EU. Seeing as the Phase II FLORA targeted patients from Ukraine, where Esbriet (pirfenidone) and Ofev (nintedanib) are not available, as well as patients waiting for reimbursement in the UK and Italy, we expect GLPG will continue to recruit such patients.

As such, if we assume that the Phase III ISABELA program will take c. 21 months to fully recruit (May-2020), 1 year to complete (May-2021), 3 months to read out (Aug-2021), 1 year to approve (Aug-2022), we are looking at a lunch in 1H'23 launch, which we have reflected in our model.

Competitive landscape: current therapies only slow the decline in lung function

IPF represents a very attractive market as there is limited competition, with Roche's Esbriet (pirfenidone) and Boehringer Ingelheim's Ofev (nintedanib) both failing to stabilise FVC and being known for their significant side effect profiles. In addition, Phase II data from Fibrogen's pamrevlumab also only slows disease progression. Aside from GLPG1690's promising efficacy and safety profiles, the once daily dosing could reflect a potential advantage over current and pipeline IPF therapies, with Esbriet being dosed three times daily after a 14-day titration period and Ofev being dosed twice daily and pamrevlumab being dosed IV every 3 weeks. GLPG1690 currently represents the only AUX inhibitor in clinical development,

while GLPG1205 is one of two drugs targeting GPR84; the other being Prometic Life Sciences' PBI-4050.

GLPG'1690 in IPF - we see an €1.0bn opportunity

- **Addressable population in IBD**: According to a paper published in the American Journal or Respiratory and Critical Care medicine in 2006, the prevalence of IPF in the US varies from 42.7 to 14.0 per 100,000 and incidence from 16.3 to 6.8 per 100,000. In 2018, we assume a patient population of c. 115k across the US and a c. 10% higher prevalence rate in the EU, with c. 128k patients. Of these patients, we assume 55% have moderate to high disease activity across all regions, with c.40% receiving treatment in the US and 30% receiving treatment in the EU.
- **Market setting & penetration**:
  Combination market: In the US, we believe Esbriet is currently capturing 33% of treated patients and Ofev is capturing around 35% of treated patients, which we expect to grow to c.35% and c.37% respectively. We assume that GLPG1690 will be added to treatment in c.3% of these patients after launch in 2023 growing to c.30% at peak penetration by 2028. In the EU, we believe Esbriet is currently capturing c.24% of treated patients with Ofev capturing c.26% of treated patients which we expect to grow to 27% and 29%, respectively. We assume that GLPG1690 will be added to treatment in 3% starting 2024 with peak penetration of 10% from 2029 and beyond.
  Monotherapy market: In the US, we see GLPG1690 capturing 5% of patients not treated with Esbriet or Ofev in 2014 growing to 60% by 2029 and beyond, given the higher unmet need in this population. In the EU, we see GLPG1690 capturing 5% of comparable patients, growing to 50% by 2029 and beyond.
- **GLPG1690 pricing**: We assume price at parity with current therapies at an annual cost of $94,200 in the US and keep the price flat going forward. In the EU, we assume a c. 60% discount to the US price ($39,600). We believe patent protection of GLPG1690 will run into the mid 2030's, beyond our forecast period which ends in 2034.
- **Peak revenues**: Based on the above assumptions, we forecast unadjusted GLPG1690 peak sales estimates of €1.0bn (€1.1bn) in IPF.
- **Valuation**: We assume that GLPG will commercialise the drug themselves outsourcing manufacturing (and so achieving a gross margin of 75%) and building a global IPF sales force of around 100 reps at peak. Despite the halt in disease progression seen for the Phase IIa FLORA study in IPF patients and the announced Phase III ISABELA program, expected to start in 2H'18, we apply a 40% probability of success to IPF sales given the small patient population of the Phase IIa FLORA trial (23 IPF patients), 17 of whom received GLPG1690 and 6 placebo) and remaining clinical trial risk. GLPG1690 sales in IPF are worth €23.2 per share / 19% of our EmV.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Table 30: Summary IPF model for 2023E-2028E

Summary Model for IPF - US	2023E	2024E	2025E	2026E	2027E	2028E
- % receiving treatment	42%	42%	42%	42%	42%	42%
Total Patients receiving treatment	26,432	26,235	25,976	25,652	25,261	24,801
Potential combination market						
Patients on Esbriet or Ofev	19,095	18,953	18,766	18,532	18,249	17,917
- GLPG1690 penetration	3%	10%	15%	20%	25%	30%
GLPG1690 patients	477	1,895	2,815	3,706	4,562	5,375
Revenue ($m)	11	179	265	349	430	506
Revenue (€m)	9	154	228	300	370	435
Potential monotherapy market						
Patients not on Esbriet or Ofev	7,336	7,282	7,210	7,120	7,011	6,884
- GLPG1690 penetration	5%	15%	30%	40%	50%	60%
GLPG1690 patients	367	1,092	2,163	2,848	3,506	4,130
Revenue ($m)	9	103	204	268	330	389
Revenue (€m)	7	88	175	231	284	335
Total US Revenue ($m)	**20**	**281**	**469**	**617**	**760**	**895**
Total US Revenue (€m)	**17**	**242**	**403**	**531**	**654**	**770**
Summary Model for IPF - EU	**2018E**	**2020E**	**2022E**	**2024E**	**2026E**	**2028E**
- % receiving treatment	30%	30%	30%	30%	30%	30%
Total Patients receiving treatment	21,599	21,593	21,549	21,465	21,343	21,181
Potential combination market						
Patients on Esbriet or Ofev	12,041	12,038	12,013	11,967	11,899	11,808
- GLPG1690 penetration	0%	3%	5%	10%	10%	10%
GLPG1690 patients	0	301	601	1,197	1,190	1,181
Revenue ($m)	0	6	24	47	47	47
Revenue (€m)	0	5	20	41	41	40
Potential monotherapy market						
Patients not on Esbriet or Ofev	9,558	9,555	9,536	9,499	9,445	9,373
- GLPG1690 penetration	0%	5%	15%	30%	40%	50%
GLPG1690 patients	0	478	1,430	2,850	3,778	4,687
Revenue ($m)	0	9	57	113	150	186
Revenue (€m)	0	8	49	97	129	160
Total EU Revenue ($m)	**0**	**15**	**80**	**160**	**197**	**232**
Total EU Revenue (€m)	**0**	**13**	**69**	**138**	**169**	**200**
Global IPF Revenue ($m)	**20**	**297**	**549**	**778**	**957**	**1,128**
Global IPF Revenue (€m)	**17**	**255**	**472**	**669**	**823**	**970**

Source: J.P. Morgan estimates

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

MOR106: Novel mechanism in the growing Atopic Dermatitis market

Drug: MOR106

Mechanism: IL-17C inhibitor

Indication: Atopic Dermatitis

Partner: Novartis

Next catalyst: Trial expansion with subcutaneous formulation in 2H'18

Peak Sales: €0.9bn (€0.5bn risk adjusted)

Risk adjustment: 50%

EmV: €4.7 (4%)

Royalty: JPMe 6-12%

MOR106 is a human IgG1 monoclonal antibody designed to selectively target IL-17C, a target discovered by GLPG with the antibody developed by MorphoSys. An intravenous (IV) formulation of MOR106 is currently in Phase II development in Atopic Dermatitis (AtD), funded by global licensing partner, Novartis as part of the July'18 license agreement. Phase Ib data for MOR106 suggests a potentially competitive profile, although the small patient population (n = 25) and lack of dose response makes the data less robust. Furthermore, IV dosing represents a commercial disadvantage; however, development of a subcutaneous (SQ) formulation is ongoing, with clinical trials expected to start in 2H'18. In our model we forecast peak unadjusted sales of €0.9bn for MOR106, and apply a 50% risk adjustment. As a result, the GLPG royalty stream is valued at €4.7/ 4% of our EmV.

Atopic Dermatitis is an underdeveloped market, with the first biologic therapy, Sanofi's Dupixent, approved in March 2017.

AtD, also called atopic eczema, is caused by skin barrier and immune system defects and characterized by very dry, red and itchy skin. It is a chronic and recurrent skin condition that often begins in early childhood, although AtD can start at any age. According to the National Eczema Association, AtD affects 18m adults (7.2%) and 9.6m (13%) children under the age of 18 in the US, of which 3.2m children (33%) have moderate to severe AtD. Furthermore, according to a paper published in the Annals of Nutrition and Metabolism in 2015, AtD affects 15-20% of children and 3% of adults worldwide, with as many as 85% of patients experiencing onset below 5 years of age.

Two of the most common tools to assess the extent and severity of atopic dermatitis in clinical trials include the Eczema Area and Severity Index (EASI) and the SCORing Atopic Dermatitis (SCORAD). The EASI score ranges between 0 (no eczema) and 72, representing a weighted score of 4 body regions considered separately; namely, the head and neck, the trunk, the upper extremities and the lower extremities. An EASI score of 7.1-21 indicates moderate disease activity, while an EASI score of 21.1-50 indicates severe disease activity and an EASI score of 50.1-72 indicates very severe disease activity.

In many cases (as much as 75%), childhood symptoms of AtD will naturally resolve as the patient moves into early adulthood, however for the patients where the disease continues there is no cure, but treatments can relieve symptoms. Some of the main treatments include:

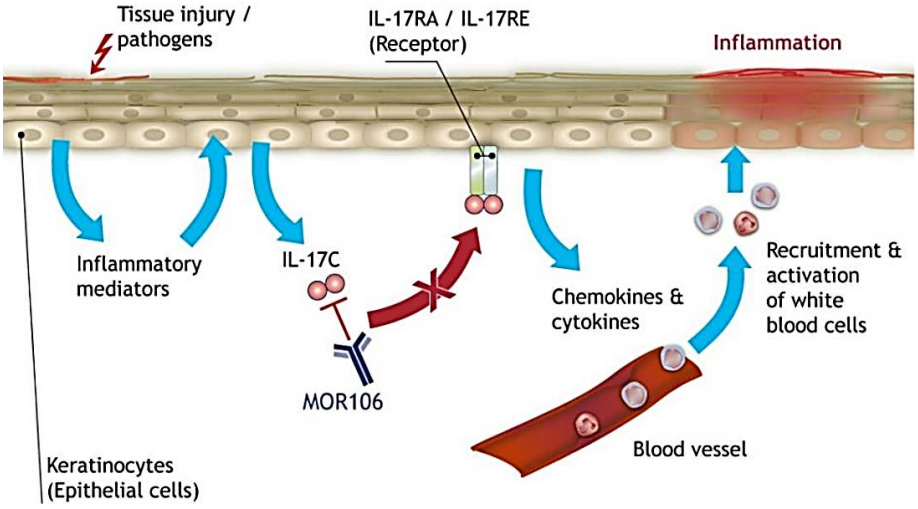
- **Moisturizers**: Applied directly to the skin to help manage dry and itchy skin.
- **Topical corticosteroids (steroids)**: Used to reduce inflammation during flare-ups. Topical corticosteroids are available in different forms and can be prescribed in different strengths from mild (e.g. hydrocortisone) to strong (e.g. mometasone).
- **Conventional synthetic DMARDs** (e.g. azathioprine, cyclosporine or MTX): Suppress the immune system to reduce inflammation and used if other treatments do not work.

- **Biologic DMARDs** (Dupixent): Sanofi / Regeneron's Dupixent (dupilumab) became the first biologic for treating adults with moderate to severe AtD following failure (or contraindication) of topical therapies and systemic immunosuppressant agents, approved by the FDA on March 28, 2017.

Mechanism of action is differentiated as it targets skin specific cytokines

IL-17C is produced mainly by epithelial cells (e.g. keratinocytes) and amplifies inflammatory mediators to induce inflamed epithelia (skin), which could make it an attractive target for drug development against dermatologic diseases. IL-17C drives skin inflammation by binding to its receptor complex, IL-17RA/IL-17RE (see Figure 18), activating multiple cytokine pathways. MOR106 is a human IgG1 monoclonal anti-IL-17C antibody that inhibits the binding of IL-17C to its receptor, IL-17RE, and thus inhibits its biological activity.

Figure 18: Role of IL-17C in skin inflammation

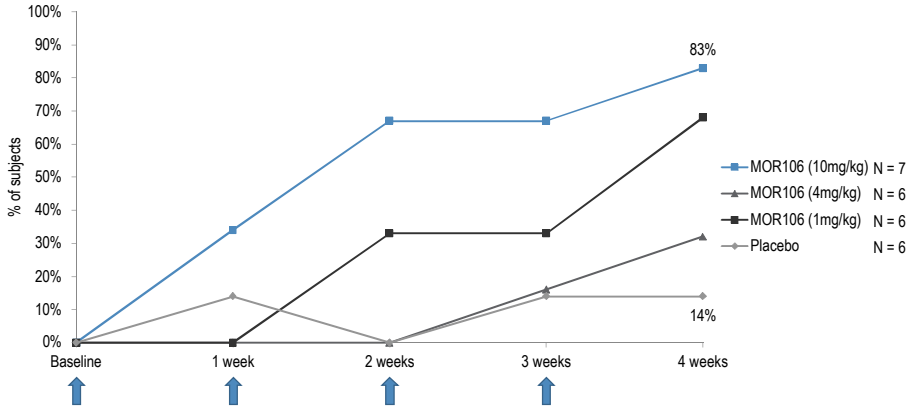


Source: Vandeghinste et al., ESDR September 2017

Initial MOR106 clinical data looks promising

On September 27, 2017, GLPG and MOR reported Phase I data evaluating multiple ascending doses (MAD) of MOR106 in patients with moderate to severe atopic dermatitis. At the highest dose (10mg/kg IV) c. 83% of MOR2016-treated patients (n = 6) reached ≥50% EASI improvement (EASI-50) by week 4 versus c. 17% of patients in the placebo arm (n = 6) (see Figure 19). This looks slightly better on a cross trial comparison vs. Dupixent (dupilumab) in the comparable Phase Ib proof of concept data in March, 2013, were Dupixent demonstrated an EASI50 of 71% for at the 300mg/qw dose (n = 21) and the placebo response was c. 19% (n = 16) at week 4. Furthermore, efficacy of MOR106 was maintained after through week 14 i.e. greater than 2 months after stopping treatment.

Figure 19: EASI-50 score with MOR106 vs. placebo



Source: Company Data

Although the signal appears similar to initial Dupixent (dupilumab) Phase Ib data, we note that the results are less robust given the smaller patient size; 25 patients in 3 cohorts of MOR106 and one placebo versus 67 patients in 3 cohorts of Dupixent (dupilumab) and one placebo. Furthermore, IV dosing of MOR106 versus SQ dosing of Dupixent (dupilumab) is a commercial disadvantage; however, management have indicated that plans to start a Phase I study evaluating a SQ formulation of MOR106 in healthy volunteers and AtD patients are ongoing.

In terms of its safety profile, MOR106 appears tolerable and safe, with all AEs categorized as mild to moderate and no infusion related reactions reported. Although the treatments emergent AEs (TEAEs) were more prevalent across the pooled MOR106-treated patients (c. 72%; n =13/18) versus placebo (c. 29%; n =2/7), we note that the AEs did not show a dose response and that the rate of discontinuation was noticeable lower for the pooled MOR106-treated patients (c. 6%; n = 1/18) versus placebo (c. 14%; n = 1/7). Management did not provide specific AEs, which limits further comparison.

MOR106's commercial agreement with Novartis and Phase II plan

On May 1, 2018, GLPG and MOR announced initiation of the IGUANA Phase II clinical trial with MOR106 in AtD patients (see Table 31). Later on July 19, 2018, GLPG and MOR signed a global licensing agreement for MOR106 with Novartis, jointly receiving an upfront payment of €95m. Additionally, GLPG and MOR will be eligible to jointly receive potential milestone payments of up to c. €850m and royalties up to low-teens to low-twenties, sharing all payments equally under the terms of their 2008 agreement. Under the terms of this licensing agreement, all future research, development, manufacturing and commercialization costs will be borne by Novartis, including the Phase II IGUANA trial and planned Phase I expansion study, evaluating the safety and efficacy of a subcutaneous (SQ) formulation of MOR106 in healthy volunteers and AtD patients. The partners will also work to identify additional indications for MOR106.

Table 31: IGUANA Phase II program in AtD

Molecule	Target	Program	Ph	Patient Population	N	Arms	Duration	Primary endpoint	Secondary endpoint	Trial Start
MOR106	IL-17C	IGUANA	II	• Adult subjects with moderate to severe atopic dermatitis • Recruitment in Europe	180	1) MOR106, 1mg/kg IV q2w or q4w 2) MOR106, 3mg/kg IV q2w or q4w 3) MOR106, 10mg/kg IV q2w 4) Placebo	12 weeks	% change in EASI score at week 12	1) % subjects with EASI-50 at 12 weeks 2) % subjects with EASI-50 at day 1 3) % subjects with EASI-50 at day 15	2Q'18

EASI = Eczema Area and Severity Index; EASI-50 = ≥50% improvement in AtD symptoms
Source: Company press releases

Competitive market & model assumptions

AtD represents an attractive market with limited competition; however, we note that oral JAK inhibitors are also in clinical development for this indication, with MOR106 lagging behind; Olumiant (baricitinib) currently in Phase III development and upadacitinib currently in Phase II development (refer back to Table 9). We currently model MOR106 having potential peak in-market sales of €0.9bn ($1.1bn), included in our model at 50% probability, with GLPG receiving royalties ranging from 6-12%. Under our current modelling, the program contributes €4.7 per share, 4% of our EmV.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

GLPG1972: Osteoarthritis represents a large opportunity but is high risk

Drug: GLPG1972

Mechanism: ADAMTS-5 inhibitor

Indication: Osteoarthritis

Partner: Servier (ex-US)

Next catalyst: Phase II ROCCELLA data, potentially in 2020

Peak Sales: €2.1bn (€0.4bn risk adjusted)

Risk adjustment: 20%

EmV: €3.3 (3%)

Royalty: US JPMe – 25%, ex-US JPMe 7-8%

GLPG1972 is disease-modifying osteoarthritis drug (DMOAD) candidate targeting a cartilage degrading enzyme called ADAMTS-5, thus inhibiting cartilage breakdown. GLPG1972 is currently in Phase II development in patients with knee osteoarthritis (OA) (see Table 32), in collaborating with Servier. Phase Ib data for GLPG1972 suggests a promising efficacy and safety profile, although clinical development risk persists given small patient size (n = 30). In our model, we forecast peak sales of €2.1bn. Given that a primary care sales force will likely be required, we expect GLPG to license out US rights to a large Pharma partner (for royalties of c.25%). Ex-US we model single digit (JPMe7/8%) royalties from GLPG's partner Servier. Given OA is a high risk development area, we apply a 20% risk adjustment to the in market sales, so GLPG1972 royalty revenues are valued at €3.3 in our EmV/ 2.9%.

Osteoarthritis background

OA, the most common form of arthritis, is a condition that is caused by breakdown of joint cartilage between bones and characterized by joint pain and stiffness, which can ultimately cause disability. OA most frequently occurs in the hands, hips and knees, with symptoms including pain, stiffness, swelling and decreased range of motion. According to Centers for Diseases Control and Prevention (CDC), OA affects more than 30m adults in the US. The risk of developing OA increases with age and is more prevalent in woman.

Although the process of OA cannot be reversed and there are no DMOADs approved to date, symptoms can usually be managed with pain relief medications (e.g. acetaminophen) and physical therapy. In cases where conservative treatments do not help, patients may consider surgery or other procedures, such as:

- **Corticosteroid injections**: Used to relieve joint pain and generally limited to 3 or 4 injections per year due to risk of medication worsening joint damage over time.
- **Osteotomy**: Surgical operation whereby a bone is cut to allow realignment.
- **Arthroplasty**: Surgical operation whereby the damaged surfaces of a joint are replaced to restore function of a joint.

Mechanism could delay cartilage breakdown

ADAMTS-5 (A Disintegrin And Metalloproteinase with ThromboSpondin-motif-5) is a key enzyme involved in cartilage degradation. More specifically, it functions to cleave aggrecan, which is the major proteoglycan component of cartilage. The cleavage of aggrecan by ADAMTS-5 results in the release of an ARGS neoepitope, which is an important biomarker of cartilage breakdown. GLPG1972 selectively inhibits ADAMTS-5, thus decreasing cartilage breakdown in treated patients, as measured by the reduction in blood levels of ARGS neoepitope.

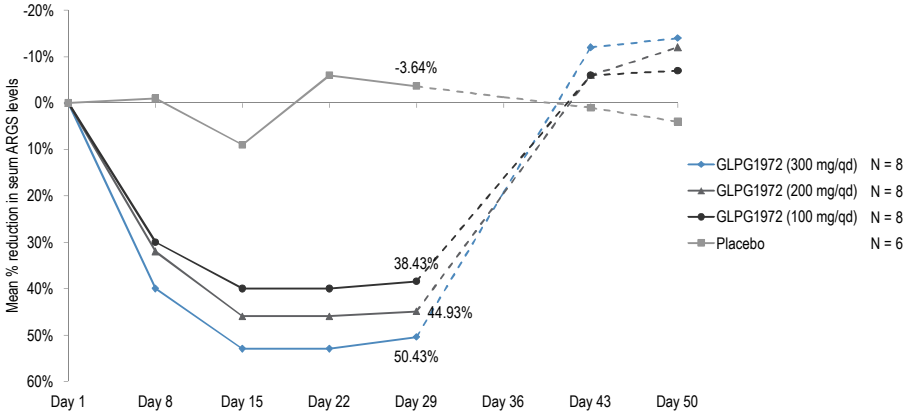
Phase I data in osteoarthritis

In Phase I, GLPG1972 demonstrated acceptable PK profile with a half-life of c. 10hrs and steady state reached in 3 days. In the multiple ascending dose (MAD)

study, healthy volunteer's serum biomarker data indicated that GLPG1972 led to a c. 50% reduction in cartilage breakdown.

On January 7, 2018, GLPG announced positive topline data from a randomized, double-bind, placebo-controlled Phase Ib study of GLPG1972 in 30 patients with knee and/or hip OA, presented at the EULAR congress in 2018. Patients were given one of three doses of GLPG1972 (100mg/qd, 200mg/qd or 300mg/qd) or placebo for 4 weeks and the primary endpoints of the study were to safety/tolerability and pharmacokinetics. GLPG1972 was reported to be well tolerated, with one discontinuation reported in the highest dose (300mg/qd) due to a reversible abnormal liver function test. The PK profile of GLPG1972 was similar to that seen in an earlier Phase I study in healthy volunteers, with a half-life of c. 10hrs and a steady state reached in 3-5 days. Furthermore, GLPG1972-treated patients achieved dose-dependent average % reductions in serum ARGS levels compared to baseline at day 29, with the greatest reduction of 50.43% in the 300mg/qd active arm versus -3.64% in the placebo arm (Figure 20).

Figure 20: Mean % reduction in serum ARGS levels with GLPG1972 vs. placebo



Source: Company Data

GLPG1972's commercial agreement with Servier and Phase II plan

GLPG and Servier initiated their strategic alliance to develop OA therapies in July, 2010. In July, 2017, Servier inlicensed the ex-US rights to GLPG1972, taking over responsibility for clinical development, registration and commercialization. Under the term of this licensing agreement, GLPG is also eligible to receive milestone payments and royalties from commercialization of products ex-US.

On June 26, 2018, GLPG and Servier announced the start of the global Phase II trial with GLPG1972 in knee OA patients (ROCCELLA) (see Table 32). As per the licensing agreement, GLPG will be responsible for ROCCELLA in the US, where 300 of a total 850 patients are targeted to be recruited, while Servier will run the trial in all other countries

Table 32: ROCCELLA Phase II program in OA

Molecule	Target	Program	Ph	Patient Population	N	Arms	Duration	Primary endpoint	Secondary endpoints	Trial Start
GLPG1972	ADAMTS-5	ROCCELLA	II	• knee osteoarthritis patients • Up to 15 countries in NA, SA, Europe and Asia (initiated in US and Hungary)	850	1) Dose A qd 2) Dose B qd 3) Dose C qd 4) Placebo	52 weeks	CFB in cartilage thickness of the cMTFC, assessed by MRI at week 52	1) Proportional of OA structural progressors* at week 52 2) CFB in WOMAC scores for pain, function & stiffness at week 52 3) CFB in knee pain, measured with 100-mm VAS at week 52	2Q'18

NA = North America; SA = South America; CFB = change from baseline; cMTFC = central medial tibiofemoral compartment; MRI = magnetic resonance imaging; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; VAS = visual analog scale
* A structural progressor is defined as a patient who had ≥8% cartilage loss in the cMTFC between baseline and week 52
Source: Company press releases

OA could be a very large market, with a c.€2.1bn in market sales opportunity for GLPG1972

With no treatment available to counteract disease progression, OA represents an important unmet medical need. We currently model GLPG1972 having potential peak in-market sales of €2.1bn ($2.4bn). GLPG have US rights, but this indication will require a large primary care sales force, which would require too much investment from GLPG to handle alone, therefore we assume GLPG license to a large pharma partner for royalties of c.25%. Servier have ex-US rights and pay single digit royalties to GLPG. In our model we apply a 20% risk adjustment to the in market sales given the high risk nature of OA development. Therefore, under our current modelling, the program contributes €3.3 per share, 3% of our EmV.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Cystic Fibrosis: No longer a key part of story, could represent a free pipeline option

Drug:
Potentiators: GLPG1837, GLPG2451 and GLPG3067
Early correctors: GLPG2222 and GLPG2851
Late correctors: GLPG2737 and GLPG3221
Triple combos: 2541+2222+2737 and 3067+2222+2737

Mechanism: CFTR modulators

Indication: Cystic Fibrosis

Partner: AbbVie

Next catalyst: Phase II FALCON data in 3Q'18

Peak Sales: not include in model

EmV: €0 (0%)

Royalty: 15-20%

GLPG's is currently running the first clinical trial using its investigational triple combo (potentiator GLPG2451, C1 corrector GLPG2222 and C2 corrector GLPG2737) in CF patients (FALCON) in collaboration with ABBV. Although, C1 corrector GLPG2222 showed signs of efficacy in the Phase II ALBATROSS study and Phase II FLAMINGO study, we remain cautious on the FALCON trial given the underwhelming results from the Phase II PELICAN study and the disclosure that GLPG and ABBV are reviewing their collaboration. That being said, we no longer see the CF franchise as being a part of GLPG's story and, as such, do not reflect it in our EmV.

Cystic Fibrosis is a hereditary lung disorder causing to a buildup of mucus in the lungs, leading to constant infections and potentially lung failure

CF is a progressive genetic disorder that causes the build-up of mucus in the lungs and other organs. This clogs the airways in the lungs and traps bacteria, leading to persistent lung infections and respiratory failure over time. According to the Cystic Fibrosis Foundation (CFF) patient registry, more than 30,000 people are living with CF in the US and more than 70,000 worldwide; with c. 1,000 new cases being diagnosed each year, 75% of which are diagnosed by age 2. The median predicted survival age is close to 40 years and symptoms include salty-tasting skin, persistent coughing, frequent lung infections, and shortness of breath. Cystic fibrosis is caused by mutations in the CFTR gene, which codes for the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) protein. Once the CFTR protein has been made in the cells, it moves to the cell surface, known as trafficking, where it functions as a chloride channel to help maintain the right salt and water balance in the lungs and other tissues. Everyone have two copies of the CFTR gene, both of which must be mutated to cause CF.

CFTR mutations are grouped into classes based on the way the mutations effect on the CFTR protein (see Table 33).

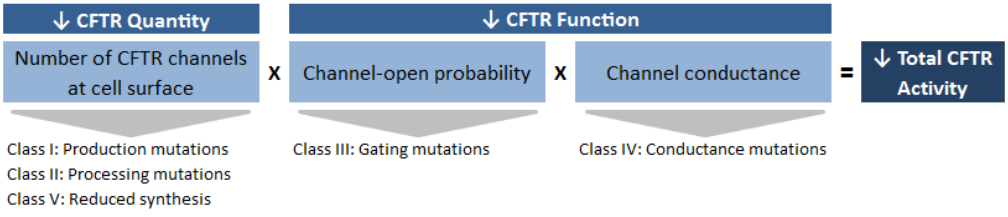
Table 33: Cystic Fibrosis Transmembrane Conductance Regulator mutation classes

	Normal	Class I	Class II	Class III	Class IV	Class V
Description	CFTR proteins is created, moves to the cell surface and allows transfer of chloride and water	No functional CFTR is created	CFTR protein is created, but misfolds, keeping it from moving to the cell surface	CFTR protein is created and moves to the cell surface, but channel gate does not open properly	CFTR protein is created and moves to the cell surface, but the function of the channel is faulty	Normal CFTR protein is created and moves to the cell surface, but in insufficient quantities
% of CF patients with ≥1 mutation		22%	88%	6%	6%	5%
Mutation examples	No mutation	G542X W1282X R553X	F508del N1303K I507del	G511D S549N	D1152H R347P R117H	3849+10kbC→T 2789+5G→A A455E
Mutation types		"Production mutations", which include nonsense mutations, some splice mutations and deletions	"Processing mutations"	"Gating mutations"	"Conduction mutations"	Includes some splice mutations
Potential therapies		Read-through compounds may allow production of full-length CFTR for nonsense mutations	Correctors such as lumacaftor or tezacaftor help defective CFTR fold correctly	Potentiators such as Kalydeco (ivacaftor) help open the CFTR channel, and also help increase the function of normal CFTR		

Source: Cystic Fibrosis Foundation

Individual CFTR mutations either decrease the quantity or function of CFTR proteins at the cell surface, resulting in reduced total CFTR activity (see Figure 21).

Figure 21: Properties of CFTR channels contributing to total activity



Source: Cystic Fibrosis Foundation

Treatment in this space is currently dominated by Vertex Pharmaceuticals (covered by US Biotechnology analyst Cory Kasimov) with their potentiator molecule Kalydeco (ivacaftor) and dual potentiator/ corrector therapies Orkambi (lumacaftor/ ivacaftor) and Symdeko (tezacaftor/ ivacaftor).

Galapagos CF platform and initial clinical data

GLPG have completed a total of five Phase II studies across their CF pipeline (see Table 34):

Table 34: GLPG's Cystic Fibrosis pipeline

	Pre-clin	Phase I	Phase II	Timeline
Potentiators				
GLPG1837				Completed Phase II SAPHIRA-1 (G551N mutation): Read out in 4Q'16 Completed Phase II SAPHIRA-2 (S1251N mutation): Read out in 4Q'16
GLPG2451				Completed Phase I safety trials
GLPG3067				Completed Phase I safety trials
Early correctors				
GLPG2222				Completed Phase II ALBATROSS (F508del/gating patients): Read out in 4Q'17 Completed Phase II FLAMINGO (F508del H/H patients): Read out in 1Q'18
GLPG2851				Entered Phase I in late 2017 in healthy volunteers
Late correctors				
GLPG2737				Completed Phase II PELICAN (F508del H/H patients): Read out in 2Q'18
GLPG3221				Entered Phase I in late 2017 in healthy volunteers
Triple Combos				
2541+2222+2737				Phase II FALCON (F508del H/H patients and het-min patients): Data expected in 3Q'18
3067+2222+2737				AbbVie has decided not to proceed with second triple combo (announced with topline results of PELICAN trial)

Source: Company data

- **SAPHIRA-2**: Tested 7 patients with the S1251N mutation [3 Kalydeco (ivacaftor) pretreated and 4 Kalydeco (ivacaftor) naïve] receiving GLPG1837 as monotherapy for 4 weeks (62 mg/bid wk1-2, 125mg/bid wk3-4). Kalydeco (ivacaftor) pretreated patients received a 1-week washout prior to treatment, with subsequent -3% decrease in ppFEV1. Treatment with GLPG1837 led to stabilization in ppFEV1 but little improvement. Kalydeco (ivacaftor) naïve patients reported a maximum ppFEV1 improvement of 5% at 4 weeks versus c. 10% as seen with Kalydeco (ivacaftor) at 24 weeks in clinical trials. Although the treatment durations are not comparable, Kalydeco (ivacaftor) data suggests that that one should see an efficacy peak at week 2.
- **SAPHIRA-1**: Tested 26 patients with the Class III G551D mutation receiving GLPG1837 as monotherapy for 4 weeks (125mg/bid wk1, 250mg/bid wk2, 500mg/bid wk3-4). 25 Kalydeco (ivacaftor) pretreated patients experienced a -5.4% decrease in ppFEV1 following a 1-week washout period prior to treatment. Treatment with GLPG1837 caused ppFEV1 levels to return to pre-washout level, while encouraging, it suggests that GLPG1837 was only as good as Kalydeco at best and so GLPG have chosen to focus on another potentiator; GLPG2451.
- **ALBATROSS**: Tested 37 patients with one copy of the Class II F508del mutation and one copy of a Class III gating mutation (F508del/gating) receiving one of two doses of GLPG2222 (150mg/qd or 300mg/qd) or placebo on top of Kalydeco (ivacaftor) for 4 weeks. GLPG2222 showed signs of efficacy when dosed on top of Kalydeco (ivacaftor), reporting a statistically significant dose dependent decrease in sweat chloride concentration, with the maximum decrease of 6.0mmol/L seen in the 300mg/qd cohort at week 4. Furthermore, mean ppFEV1 levels increased by 2.2% in the 300mg/qd cohort at week 4. In terms of safety profile, GLPG2222 was reported as being well tolerated, with no serious adverse events and no adverse events leading to discontinuations.
- **FLAMINGO**: Tested 59 F508 H/H patients receiving GLPG2222 as monotherapy (dose A, B, C or D) or placebo for 4 weeks. GLPG2222 showed signs of efficacy when dosed as monotherapy, reporting a statistically significant dose dependent decrease in sweat chloride concentration, with the maximum decrease of -18.3mmol/L seen in the Dose C cohort at week 4. On terms of safety profile, GLPG2222 was again reported as being well tolerated, with a total of four SAEs reported in three patients, two in the placebo arm (n = 11) and one in the Dose B cohort (n = 10), and no adverse events leading to discontinuations

- **PELICAN**: Tested 22 F508del H/H patients on Orkambi (lumacaftor/ivacaftor) receiving GLPG2737 (n=14) or placebo (n = 8) for 4 weeks. The combination only demonstrated a ppFEV1 improvement of 3.4% (p=0.08) vs. placebo compared to differences (non-placebo adjusted) of c. 7-11% seen when adding VRTX's own pipeline correctors to Symdeco (tezacaftor/ivacaftor).

We do not include any value for the CF portfolio in our model

Table 35 summarizes an overview of the disease modulating therapies approved or under investigation for CF excluding the majority of anti-inflammatory/anti-infective therapies. VRTX currently has the only marketed drugs for CF that treat the underlying cause of the disease and based on the triple combo data we have seen to date, VRTX is setting the bar high. Given data seen to date on the GLPG portfolio and factoring in Vertex's commanding commercial and clinical position (they expect filing of their first triple combination no later than mid-19) we do not ascribe any value to the CF programme in our model – but it remains a free pipeline option, provided AbbVie and GLPG can come to an agreement on the future of the collaboration.

Table 35: Cystic Fibrosis competitive landscape

Company	Product(s)	Mechanism	Stage	Notes
Active				
Vertex	Kalydeco (ivacaftor) Orkambi (lumacaftor/ivacaftor) Symdeko (tezacaftor/ivacaftor) VX-440, VX-152 VX-659, VX-445	CFTR modulators	Approved for gating mutations ≥2yrs Approved for F508 H/H ≥6yrs Approved in residual function patients ≥12yrs Phase II Phase III	Pivotal Phase III triple combo trials with VX-659/445 + Symdeko underway in F508del H/H and het-min patients. Initial results Initial results expected in 1H'19, With NDA submission anticipated no later than mid-2019
Concert Pharma	CTP-656 (Now VX-561)	CFTR modulator	Phase II	Purchased by VRTX for use in triple combinations
ProQR Therapeutics	QR-010	RNA Repair	Phase II	Positive biomarker data reported, no clinical efficacy results as yet
Proteostasis Therapeutics	PTI-428, PTI-801, PTI-808, PTI-NC-733	CFTR modulators	Phase II, Phase I	Proof-of-concept Triple combo study of 428+801+808 is underway. Initial results expected in 2H'18
Celtaxxys	Acebilustat	LTA4H inhibitor	Phase II	Anti-inflammatory adjunct to current CF regimens, aims to prevent long term pulmonary decline
Corbus	Resunab	Binds to CB2 receptors	Phase II	Anti-inflammatory adjunct to current CF regimens, the company will likely focus on reducing acute pulmonary exacerbations

Source: J.P. Morgan Research

Partnership with AbbVie and Phase II CF triple study

GLPG and ABBV entered into a global alliance in September 2013, with the aim of developing a triple combination therapy to address 90% of CF patients worldwide. In April, 2018, GLPG announced the start of its first clinical trial with a triple combo. The Phase II FALCON study investigates GLPG's first triple combo (potentiator GLPG2451, C1 corrector GLPG2222 and C2 corrector GLPG2737) in CF patients with 1.) two copies of the Class II F508del mutation (F508del H/H) and 2.) patients with one copy of the F508del mutation and one copy of a mutation which results in minimal CFTR function (F508del het-min). The trial is composed of two parts; Part one involves 8 F508del H/H patients treated with a lower dose, while part two involves F508del H/H and F508del het-min patients treated with a higher dose (Table 36). Based on the underwhelming Phase II topline results of GLPG2737 in F508del H/H patients treated with Orkambi (lumacaftor/ivacaftor) (PELICAN) in

June, 2018, ABBV decided not to proceed with the second triple combo (potentiator GLPG3067, C1 corrector GLPG2222 and C2 corrector GLPG2737).

The Phase II FALCON trial represents the next catalyst for GLPG's CF franchise, with topline data from part 1 (low dose in F508del H/H patients) expected to read out in 3Q'18 (Table 36). Given the underwhelming results from the Phase II PELICAN study and the disclosure that GLPG and ABBV are reviewing their collaboration, our expectations for the FALCON trial are low. Furthermore, we note that the data from part 1 will only involve the lower dose (exact dose undisclosed), and as such, we do not expect a very potent signal. While we include no value for the CF portfolio in our model, we still expect that underwhelming FALCON data could still lead to the shares underperforming a couple of percent given the loss of some pipeline optionality. However, while this would be a sentiment based move, we include no fundamental value for the CF portfolio.

Table 36: Phase II FALCON protocol design

Molecule	MoA	Program	Ph	Patient Population	N	Arms	Duration	Primary endpoints	Secondary endpoint	Read out
GLPG2451+ GLPG2222+ GLPG2737	CFTR modulators	FALCON (part 1)	II	• Adult CF patients • Multiple centers in Europe (initiated in the UK)	8	Dose A Dual combination (2451+ 2222) for 2 week, followed by triple combination (2451+2222+2737) for 2 weeks in F508del H/H patient	4 weeks	1) Safety 2) Tolerability 3) PK	1) CFB in [sweat chloride] 2) CFB in ppFEV1	3Q'18
GLPG2451+ GLPG2222+ GLPG2737	CFTR modulators	FALCON (part 2)	II	• Adult CF patients • Multiple centers in Europe (initiated in the UK)	16	1) Dose B Dual combination (2451+ 2222) for 2 week, followed by triple combination (2451+2222+2737) for 2 weeks in F508del H/H patient 2) Dose B Dual combination (2451+ 2222) for 2 week, followed by triple combination (2451+2222+2737) for 2 weeks in F508del het-min patient	4 weeks	1) Safety 2) Tolerability 3) PK	1) CFB in [sweat chloride] 2) CFB in ppFEV1	2019

F508 H/H patients = patients with two copies of the Class II F508del mutation; F508 het-min patients = patients with one copy of the Class II F508del mutation and one copy of a mutation that results in minimal CFTR function; PK = pharmacokinetics; CFB = change from baseline; ppFEV1 = percent predicted forced expiratory volume in 1 second
Source: Company press releases, Clinicaltrials.gov

Management Profile

Executive Team

Onno van de Stolpe: Chief Executive Officer

Mr. Van de Stolpe founded GLPG in 1999 and has served as Chief Executive Officer and a member of their board of directors since 1999. Prior to GLPG, he was the Managing Director of Genomics at IntroGene B.V. between 1998 and 1999. Mr. Van de Stolpe started his career as Manager of Business Development at MOGEN International N.V. in Leiden, where after he was responsible for recruiting biotechnology and medical device companies to the Netherlands at the Netherlands Foreign Investment Agency in California. He then joined Molecular Probes, Inc. in the United States in 1994, where he established the European headquarters of Molecular Probes, Inc. in 1995 and served as the Managing Director of Molecular Probes Europe. Mr. Van de Stolpe is a member of the supervisory board of the Stichting Institute for Human Organ and Disease Model Technologies and holds an MSc degree from Wageningen University.

Piet Wigerinck: Chief Scientific Officer

Dr. Piet Wigerinck, Ph.D. joined GLPG in April 2008 with over 25 years of research and development experience and served as its Senior Vice President of Development until his appointment as Chief Scientific Officer in April 2012. In this role, Dr. Wigerinck oversees the development of GLPG's drug candidates, including pre-clinical development, chemistry manufacturing and control (CM&C), regulatory aspects and clinical development. Prior to GLPG, Dr. Wigerinck was Vice President of Drug Discovery, Early Development and CM&C, and a member of the Management Board at Tibotec-Virco Comm. VA (a subsidiary of Johnson & Johnson). Dr. Wigerinck started his professional career as a medicinal chemist at Janssen Research Foundation in 1992 and later joined Tibotec Group NV in 1998, where he played a key role in Tibotec's expansion into novel diseases such as Hepatitis C and advanced several compounds into clinical trials, including TMC114 (Prezista™) and TMC435 (Olysio™). Dr. Wigerinck is inventor on more than 25 patent applications and holds a Ph.D. from the K.U. Leuven.

Bart Filius: Chief Operating Officer & Chief Financial Officer

Mr. Bart Filius, MBA, joined GLPG in December 2014 as Chief Financial Officer and was additionally appointed as its Chief Operational Officer in September 2017. Prior to GLPG, Mr. Filius worked at Sanofi S.A., for over 13 years. During his time at Sanofi, Mr. Filius served as Vice President for Mergers & Acquisitions and CFO and Country Manager of Sanofi in the Netherlands before he was appointed Chief Financial Officer of Sanofi Europe during the last three years. Prior to Sanofi, he was a strategy consultant at Arthur D. Little. Mr. Filius holds an MBA degree from INSEAD and a BSc degree in business from Nyenrode University.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Board of Directors

Raj Parekh: Chairman of the Board

Dr. Parekh, MA, DPhil, and has served as Chairman of the board of directors since 2004. He started his professional career in tandem with his academic career at the University of Oxford by co-founding Oxford GlycoSciences PLC, where he served as Chief Scientific Officer and Chief Executive Officer from 1988 until its sale to Celltech Group PLC in 2003. Dr. Parekh has founded or served on the boards of several life sciences companies in the United States and Europe including Celldex Therapeutics, Inc.; Avila Therapeutics, Inc.; EUSA Pharma (Europe) Limited; Thiakis Limited; and Amsterdam Molecular Therapeutics (AMT) Holding N.V. (now uniQure). Dr. Parekh is a General Partner at Advent Life Sciences LLP, which he joined in 2005, and currently serves as a member of the board of directors of Advent Venture Partners; Advent Life Sciences LLP; Aleta Inc.; Arrakis, Inc.; Aura Inc.; Artax Inc.; Capella BioSciences Ltd.; Cellnovo Limited; EnCipher Limited; Itara Limited; Levicept Limited; Macrolide Pharmaceuticals, Inc.; PE Limited; and Project Paradise Limited. He is also a member of the supervisory board of the Novartis Venture Fund. Dr. Parekh holds an MA in Biochemistry and DPhil in Molecular Medicine from the University of Oxford, where he was a Senior Research Fellow and Professor.

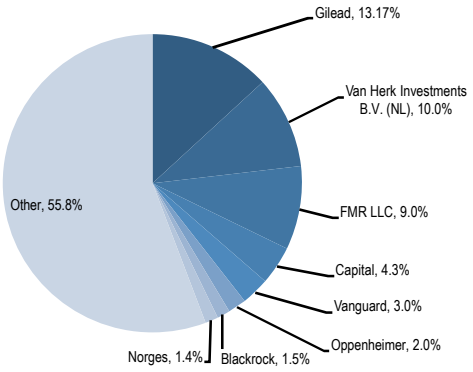
James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Appendix

Shareholders

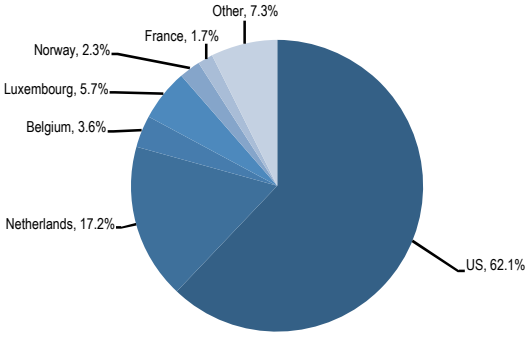
As of March 31, 2018, the total number of issued shares was 51.2 million with a free float of 77.1%. The number of investors is fairly diverse with Gilead being the largest investor with 13.2% stake in the company followed by Fidelity Management and Research (FMR) with 9.0% and Van Herk Investments with 8.7%. In addition, management and insiders own 1.1% of the company, with CEO Onno Van de Stolpe owning c.1%.

Figure 22: Top 8 shareholders and by investor type



Source: Bloomberg

Figure 23: Geographic split of shareholders



Source: Bloomberg

Intellectual Property

Galapagos' proprietary development technology Filgotinib has a combination of matter patent until 2034, with the possibility for an extension of up to 5 years.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Investment Thesis, Valuation and Risks

Galapagos *(Overweight; Price Target: €120.00)*

Investment Thesis

We initiate coverage on Galapagos with an Overweight rating and a Jun-19 PT of €120 per share/ $140 per ADR, indicating c.15% upside potential from current levels. The key value is the JAK-1 specific inhibitor filgotinib (partnered with Gilead) in autoimmune diseases, which has an efficacy profile at least as good as other (less selective) members of the JAK inhibitor class and a best in class safety profile - which we believe will drive uptake of the drug from launch in 2020. We forecast peak in market filgotinib sales of €4.4bn and include €3.2bn in our model after applying risk adjustments. In addition we include value from GLPG1690 which, based on Phase II data, could be the first asset in Idiopathic Pulmonary Fibrosis to halt disease progression and we forecast peak sales of €1bn and include €0.4bn in our model. GLPG have full commercial rights to GLPG1690 and intend to commercialise the asset worldwide. We also include value for royalties from MOR106 in atopic dermatitis (partnered with Novartis) and GLPG1972 in Osteoarthritis. We do not include any value in our model for Cystic Fibrosis given Vertex's commanding position and fairly underwhelming data presented to date, so any positive surprise in this programme could represent a free pipeline option.

Valuation

We value Galapagos using our Embedded Value methodology (product by product NPV analysis), which results in our Jun-19 price target of €120 per share for the GLPG share and $140 for the GLPG ADR. In our EmV we include €82.1 per share relating to royalties, profit share and related commercial and R&D costs of filgotinib in autoimmune indicaitons. From the other pipeline we include €23.2 per share for GLPG1690 in Idiopathic Pulmonary Fibrosis, €4.7 per share for MOR106 in Atopic Dermatitis and €3.3 per share for GLPG1972 in Osetoarthritis. We do not include any value for the Cystic Fibrosis portfolio. We include cash of €1.2bn, being €21.6 per share. Offsetting this, we include SG&A costs of -€7.8 per share, R&D of -€7 per share and Capex of -€1 per share. This leads to an EmV of €119 per share, which informs our Jun-19 PT of €120 per share. For the ADR, we translate our GLPG NV value into USD using a €:$ FX rate of 1.16, giving $140.

Risks to Rating and Price Target

- Clinical trial risk relating to the remaining trials of the FINCH programme (FINCH 1 & 3) and the ISABELA programme in IPF.
- The key markets for filgotinib (RA, CD, UC, PsA and AS) are competitive, with the potential for additional competition within the JAK class from ABBV's upadacitinib, this could impact the commercial potential of filgotinib.
- If the MANTA testicular safety study demonstrates an impact of filgotinib on lowering sperm counts, this could lead to a warning on the label, which could reduce the commercial potential in some indications.
- Galapagos has no experience in commercialising assets, therefore it may not be able to extract the full value from GLPG1690 by leading the worldwide commercialisation in IPF.
- The same risks apply to the Galapagos ADR.

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Galapagos: Summary of Financials

Income Statement	FY16A	FY17A	FY18E	FY19E	FY20E
Revenue	**152**	**156**	**251**	**277**	**259**
Gross profit	**152**	**156**	**251**	**277**	**259**
SG&A	(24)	(27)	(36)	(51)	(62)
R&D expenses	(140)	(219)	(316)	(362)	(346)
Reported EBITDA	**(7)**	**(86)**	**(94)**	**(130)**	**(142)**
Adj. EBITDA	**(7)**	**(86)**	**(94)**	**(130)**	**(142)**
D&A	(4)	(4)	(7)	(6)	(7)
Adj. EBIT	**(11)**	**(90)**	**(101)**	**(136)**	**(149)**
Net Interest	8	(26)	7	6	5
Adj. PBT	**54**	**(116)**	**(95)**	**(130)**	**(144)**
Tax	(0)	(0)	(0)	0	0
Minority Interest	-	-	-	-	-
Adj. Net Income	**54**	**(116)**	**(95)**	**(130)**	**(144)**
Reported EPS	1.14	(2.34)	(1.85)	(2.54)	(2.82)
Adj. EPS	**1.14**	**(2.34)**	**(1.85)**	**(2.54)**	**(2.82)**
DPS	**-**	**-**	**-**	**-**	**-**
Payout ratio	-	-	-	-	-
Shares outstanding	46	49	51	51	51

Cash Flow Statement	FY16A	FY17A	FY18E	FY19E	FY20E
Cash flow from operating activities	**239**	**(147)**	**(189)**	**(198)**	**(188)**
o/w Depreciation & amortization	3	4	4	4	5
o/w Changes in working capital	240	(148)	(195)	(205)	(193)
Cash flow from investing activities	**(7)**	**(1)**	**(7)**	**(7)**	**(7)**
o/w Capital expenditure	(4)	(5)	(5)	(5)	(5)
as % of sales	*2.9%*	*3.4%*	*2.1%*	*1.9%*	*2.1%*
Cash flow from financing activities	**396**	**353**	**0**	**0**	**0**
o/w Dividends paid	-	-	-	-	-
o/w Shares issued/(repurchased)	396	353	0	0	0
o/w Net debt issued/(repaid)	(0)	(0)	0	0	0
Net change in cash	**633**	**178**	**(196)**	**(206)**	**(195)**
Adj. Free cash flow to firm	**243**	**(152)**	**(194)**	**(204)**	**(193)**
y/y Growth	*(302.2%)*	*(162.6%)*	*27.2%*	*5.1%*	*(5.2%)*

Balance Sheet	FY16A	FY17A	FY18E	FY19E	FY20E
Cash and cash equivalents	973	1,151	955	750	555
Accounts receivable	20	40	62	67	63
Inventories	0	0	0	0	0
Other current assets	14	6	6	6	6
Current assets	**1,007**	**1,198**	**1,024**	**824**	**625**
PP&E	15	17	18	20	20
Intangible assets	1	2	1	1	1
LT investments	-	-	-	-	-
Other non current assets	61	72	70	70	70
Total assets	**1,083**	**1,286**	**1,113**	**914**	**716**
Short term borrowings	0	0	0	0	0
Payables	31	47	53	62	61
Other short term liabilities	72	125	99	74	35
Current liabilities	**104**	**172**	**152**	**136**	**96**
Long-term debt	0	0	0	0	0
Pension liabilities	4	4	4	4	4
Other long term liabilities	221	103	110	38	5
Total liabilities	**325**	**274**	**262**	**174**	**101**
Shareholders' equity	759	1,012	851	740	615
Minority interests	-	-	-	-	-
Total liabilities & equity	**1,083**	**1,286**	**1,113**	**914**	**716**
BVPS	**16.60**	**20.45**	**16.64**	**14.46**	**12.02**
y/y Growth	*62.4%*	*23.2%*	*(18.6%)*	*(13.1%)*	*(16.9%)*
Net debt/(cash)	(973)	(1,151)	(955)	(750)	(555)

Ratio Analysis	FY16A	FY17A	FY18E	FY19E	FY20E
Gross margin	100.0%	100.0%	100.0%	100.0%	100.0%
SG&A/Sales	15.5%	17.5%	14.4%	18.5%	23.9%
R&D/Sales	92.1%	140.1%	125.8%	130.7%	133.8%
Adj. EBITDA margin	(4.8%)	(54.8%)	(37.4%)	(47.0%)	(55.1%)
Adj.EBIT margin	(7.6%)	(57.6%)	(40.2%)	(49.2%)	(57.7%)
Tax rate	0.4%	(0.2%)	(0.1%)	0.0%	0.0%
Net profit margin	35.6%	(74.2%)	(37.7%)	(46.9%)	(55.8%)
ROE	9.6%	(13.1%)	(10.2%)	(16.3%)	(21.3%)
ROA	7.1%	(9.8%)	(7.9%)	(12.8%)	(17.7%)
ROCE	(2.0%)	(10.2%)	(10.9%)	(17.1%)	(22.0%)
Net debt/Equity	(128.3%)	(113.8%)	(112.2%)	(101.4%)	(90.2%)
Net debt/EBITDA	13315.5%	1346.2%	1016.9%	576.2%	389.4%
Sales/Assets (x)	0.2	0.1	0.2	0.3	0.3
Assets/Equity (x)	1.4	1.3	1.3	1.3	1.2
Interest cover (x)	-	NM	14.4	20.4	29.4
Operating leverage	(58.0%)	23998.5%	20.5%	341.6%	(140.9%)
Revenue y/y Growth	150.3%	2.8%	61.3%	10.2%	(6.7%)
Adj. EBITDA y/y Growth	(91.5%)	1070.1%	9.9%	38.5%	9.5%
Adj. EPS y/y Growth	(134.4%)	(304.8%)	(20.8%)	37.2%	11.1%

Valuation	FY16A	FY17A	FY18E	FY19E	FY20E
Adj. P/E (x)	91.7	NM	NM	NM	NM
Reported P/E (x)	91.7	NM	NM	NM	NM
P/BV (x)	6.3	5.1	6.3	7.2	8.7
EV/EBITDA (x)	NM	NM	NM	NM	NM
Dividend yield	-	-	-	-	-
FCFF yield	4.9%	(2.9%)	(3.6%)	(3.8%)	(3.6%)

Source: Company reports and J.P. Morgan estimates.
Note: € in millions (except per-share data).Fiscal year ends Dec. o/w - out of which

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

Galapagos ADR: Summary of Financials

Income Statement	FY16A	FY17A	FY18E	FY19E	FY20E
Revenue	**152**	**156**	**251**	**277**	**259**
Gross profit	**152**	**156**	**251**	**277**	**259**
SG&A	(24)	(27)	(36)	(51)	(62)
R&D expenses	(140)	(219)	(316)	(362)	(346)
Reported EBITDA	**(7)**	**(86)**	**(94)**	**(130)**	**(142)**
Adj. EBITDA	**(7)**	**(86)**	**(94)**	**(130)**	**(142)**
D&A	(4)	(4)	(7)	(6)	(7)
Adj. EBIT	**(11)**	**(90)**	**(101)**	**(136)**	**(149)**
Net Interest	8	(26)	7	6	5
Adj. PBT	**54**	**(116)**	**(95)**	**(130)**	**(144)**
Tax	(0)	(0)	(0)	0	0
Minority Interest	-	-	-	-	-
Adj. Net Income	**54**	**(116)**	**(95)**	**(130)**	**(144)**
Reported EPS	1.14	(2.34)	(1.85)	(2.54)	(2.82)
Adj. EPS	**1.14**	**(2.34)**	**(1.85)**	**(2.54)**	**(2.82)**
DPS	**-**	**-**	**-**	**-**	**-**
Payout ratio	-	-	-	-	-
Shares outstanding	46	49	51	51	51

Cash Flow Statement	FY16A	FY17A	FY18E	FY19E	FY20E
Cash flow from operating activities	**239**	**(147)**	**(189)**	**(198)**	**(188)**
o/w Depreciation & amortization	3	4	4	4	5
o/w Changes in working capital	240	(148)	(195)	(205)	(193)
Cash flow from investing activities	**(7)**	**(1)**	**(7)**	**(7)**	**(7)**
o/w Capital expenditure	(4)	(5)	(5)	(5)	(5)
as % of sales	*2.9%*	*3.4%*	*2.1%*	*1.9%*	*2.1%*
Cash flow from financing activities	**396**	**353**	**0**	**0**	**0**
o/w Dividends paid	-	-	-	-	-
o/w Shares issued/(repurchased)	396	353	0	0	0
o/w Net debt issued/(repaid)	(0)	(0)	0	0	0
Net change in cash	**633**	**178**	**(196)**	**(206)**	**(195)**
Adj. Free cash flow to firm	**235**	**(152)**	**(194)**	**(204)**	**(193)**
y/y Growth	*(294.7%)*	*(164.8%)*	*27.2%*	*5.1%*	*(5.2%)*

Balance Sheet	FY16A	FY17A	FY18E	FY19E	FY20E
Cash and cash equivalents	973	1,151	955	750	555
Accounts receivable	20	40	62	67	63
Inventories	0	0	0	0	0
Other current assets	14	6	6	6	6
Current assets	**1,007**	**1,198**	**1,024**	**824**	**625**
PP&E	15	17	18	20	20
Intangible assets	1	2	1	1	1
LT investments	-	-	-	-	-
Other non current assets	61	72	70	70	70
Total assets	**1,083**	**1,286**	**1,113**	**914**	**716**
Short term borrowings	0	0	0	0	0
Payables	31	47	53	62	61
Other short term liabilities	72	125	99	74	35
Current liabilities	**104**	**172**	**152**	**136**	**96**
Long-term debt	0	0	0	0	0
Pension liabilities	4	4	4	4	4
Other long term liabilities	221	103	110	38	5
Total liabilities	**325**	**274**	**262**	**174**	**101**
Shareholders' equity	759	1,012	851	740	615
Minority interests	-	-	-	-	-
Total liabilities & equity	**1,083**	**1,286**	**1,113**	**914**	**716**
BVPS	**16.60**	**20.45**	**16.64**	**14.46**	**12.02**
y/y Growth	*62.4%*	*23.2%*	*(18.6%)*	*(13.1%)*	*(16.9%)*
Net debt/(cash)	(973)	(1,151)	(955)	(750)	(555)

Ratio Analysis	FY16A	FY17A	FY18E	FY19E	FY20E
Gross margin	100.0%	100.0%	100.0%	100.0%	100.0%
SG&A/Sales	15.5%	17.5%	14.4%	18.5%	23.9%
R&D/Sales	92.1%	140.1%	125.8%	130.7%	133.8%
Adj. EBITDA margin	(4.8%)	(54.8%)	(37.4%)	(47.0%)	(55.1%)
Adj.EBIT margin	(7.6%)	(57.6%)	(40.2%)	(49.2%)	(57.7%)
Tax rate	0.4%	(0.2%)	(0.1%)	0.0%	0.0%
Net profit margin	35.6%	(74.2%)	(37.7%)	(46.9%)	(55.8%)
ROE	9.6%	(13.1%)	(10.2%)	(16.3%)	(21.3%)
ROA	7.1%	(9.8%)	(7.9%)	(12.8%)	(17.7%)
ROCE	(2.0%)	(10.2%)	(10.9%)	(17.1%)	(22.0%)
Net debt/Equity	(128.3%)	(113.8%)	(112.2%)	(101.4%)	(90.2%)
Net debt/EBITDA	13315.5%	1346.2%	1016.9%	576.2%	389.4%
Sales/Assets (x)	0.2	0.1	0.2	0.3	0.3
Assets/Equity (x)	1.4	1.3	1.3	1.3	1.2
Interest cover (x)	0.9	NM	14.4	20.4	29.4
Operating leverage	(58.0%)	23998.5%	20.5%	341.6%	(140.9%)
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Dividend yield	-	-	-	-	-
FCFF yield	4.8%	(3.0%)	(3.7%)	(3.9%)	(3.7%)

Source: Company reports and J.P. Morgan estimates.
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James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

James P Quigley
(44-20) 7742-1444
james.quigley@jpmorgan.com

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Galapagos (GLPG.AS, GLPG NA) Price Chart

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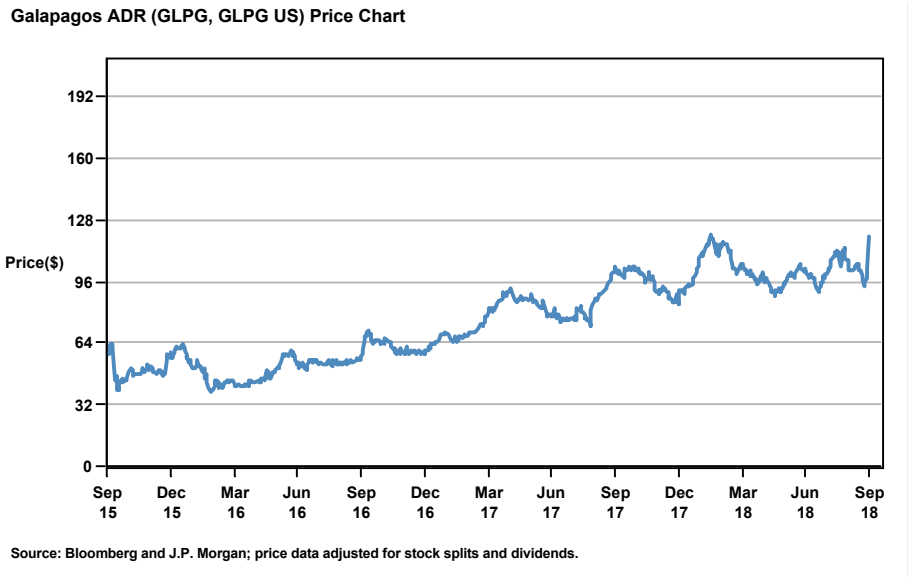

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