Pamrevlumab was well tolerated in a Phase I study in IPF. Study 002 was a Phase I open-label study to determine the safety and pharmacokinetics of escalating single doses of pamrevlumab. Patients with a diagnosis of IPF by clinical features and surgical lung biopsy received a single IV dose of pamrevlumab at 1, 3, or 10 mg/kg. A total of 21 patients were enrolled in the study; six patients received a dose of 1 mg/kg, nine patients received 3 mg/kg, and six patients received 10 mg/kg. Pamrevlumab was well tolerated across the range of doses studied; and there were no dose-limiting toxicities. TEAE that were considered to be possibly related by the principal investigator to pamrevlumab were mild and self-limited, consisting of pyrexia, cough and headache.

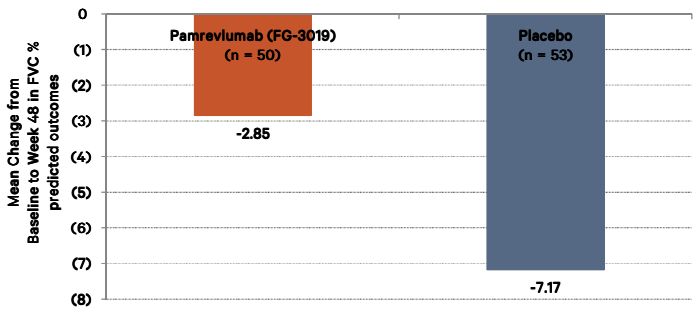
Pamrevlumab generated compelling Phase II data in the PRAISE study

In August 2017, FibroGen reported positive top-line results from a Phase II study. The randomized, double-blind, placebo-controlled study (PRAISE) was designed to evaluate the safety and efficacy of pamrevlumab in patients with mild-to-moderate IPF (baseline FVC percentage predicted of 55%), and top-line results from two sub-studies that were added to evaluate the safety of combining pamrevlumab with pirfenidone and nintedanib.

In the double-blind, placebo-controlled 48-week portion of this study, 103 patients were randomized (1:1) to receive either 30mg/kg of pamrevlumab or placebo intravenously every three weeks. Lung function assessments were conducted at baseline and at Weeks 12, 24, 36 and 48. Quantitative HRCT assessments were performed at baseline and on Weeks 24 and 48.

Pamrevlumab met the primary efficacy endpoint of change of FVC percent predicted. The average decline (least squares mean) in FVC percent predicted from baseline to Week 48 was 2.85 in the pamrevlumab arm (n=50) as compared to an average decline of 7.17 in the placebo arm (n=51), which was a statistically significant difference of 4.33 (p=0.0331, using a linear slope analysis in the Intent to Treat (ITT) population).

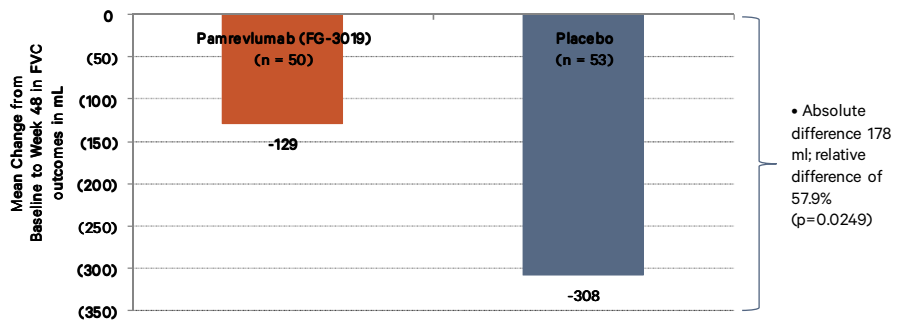
Exhibit 33: Pamrevlumab met the primary endpoint in PRAISE



Source: Company filings, Berenberg Capital Markets

Pamrevlumab-treated patients had an average decrease (least squares mean) in FVC of 129 ml at week 48 compared to an average decrease of 308 ml in patients receiving placebo, a statistically significant difference of 178 ml (p=0.0249, using a linear slope analysis in the ITT population). This represents a 57.9% relative difference.

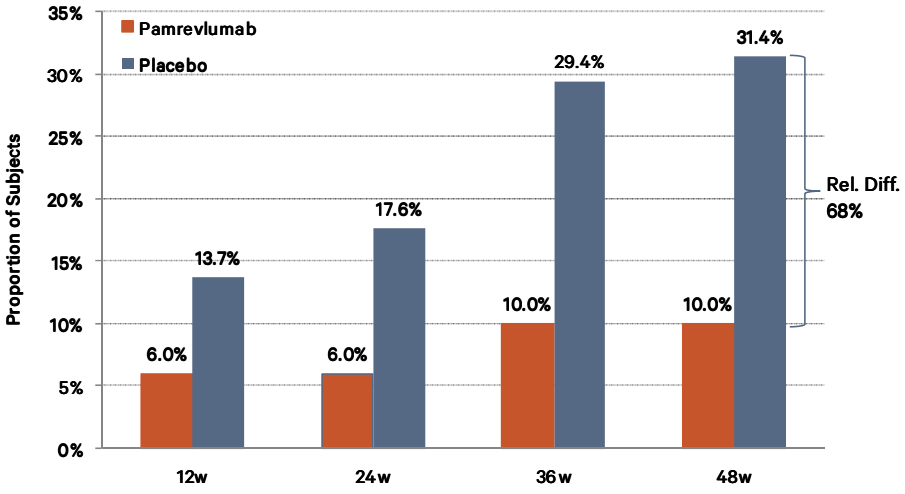
Exhibit 34: Pamrevlumab generated a placebo-adjusted improvement in FVC of 178 mL



Source: Company filings, Berenberg Capital Markets

The pamrevlumab-treated arm had a lower proportion of patients (10%) who experienced disease progression (defined by a decline in FVC percent predicted of greater than or equal to 10%) or death, than did the placebo arm (31.4%) at week 48 (p=0.0103). The percentage of pamrevlumab patients who experienced disease progression and discontinued therapy was less than 15% of that in the placebo arm.

Exhibit 35: Pamrevlumab demonstrated an improvement in disease progression



	12w	24w	36w	48w
p-value	0.1235	0.0527	0.0172	0.0103
pamrevlumab, n (%)	3 (6%)	3 (6%)	5 (10%)	5 (10%)
placebo, n (%)	7 (13.7%)	9 (17.6%)	15 (29.4%)	16 (31.4%)
relative diff	-56.0%	-66.0%	-66.0%	-68.0%

Source: Company filings, Berenberg Capital Markets

Notably, the pamrevlumab arm achieved a statistically significant reduction in the rate of progression of lung fibrosis compared to placebo using HRCT to measure quantitative lung fibrosis (QLF). The change in QLF volume from baseline to Week 24 for pamrevlumab-treated patients was 24.8 ml vs. 86.4 ml for placebo, with a treatment difference of -61.6 ml, p=0.009. The change in QLF volume from baseline to 48 weeks was 75.4 ml in pamrevlumab-treated patients vs. 151.5 ml in patients on placebo, with a treatment difference of -76.2 ml, p=0.038. Neither FibroGen nor we are aware of any other IPF therapies that have shown a statistically significant effect on lung fibrosis as measured by quantitative HRCT analysis.

Pamrevlumab Phase III (ZEPHYRUS) began enrolling on July 22, 2019

FibroGen recently began enrolling ZEPHYRUS, a double-blind, placebo-controlled Phase III trial of pamrevlumab in approximately 565 IPF patients. The study design is anticipated to be similar to the PRAISE Phase IIb study; it is powered to meet the FDA requirement of a highly statistically-significant result in the primary efficacy endpoint of change from baseline in FVC, according to FibroGen. Secondary endpoints include a composite clinical outcome of disease progression, patient reported outcomes, and quantitative changes in lung fibrosis volume from baseline, among others.

Notably, unlike in Galapagos/Gilead's Phase III ISABELA program, FibroGen is enrolling IPF patients who are **not** being treated with approved therapies such as pirfenidone and nintedanib. The primary completion for ZEPHYRUS is listed as March 2023.

Biogen's Phase IIb study (SPIRIT) was recently halted

BG00011 (formerly STX-100) is a first-in-class humanized monoclonal antibody targeted against the integrin αvβ6. As noted earlier in this report, TGF-β is a critical pro-fibrotic growth factor that is believed to play a critical role in the development of fibrosis. The αvβ6 integrin functions as an activator of TGF-β. In mice, inhibition of αvβ6 attenuates the development of bleomycin-induced fibrosis. Biogen began conducting a Phase IIb study in H218, however, **one of the KOLs we spoke to mentioned that Biogen recently halted the study. We confirmed that the study was halted with Galapagos.** Galapagos does not know

why the study was halted. We did not find any further information online. The KOL suspects the issue could be biology and/or trial design-related; see the KOL View section later on in this report for further details. See here for more details regarding SPIRIT.

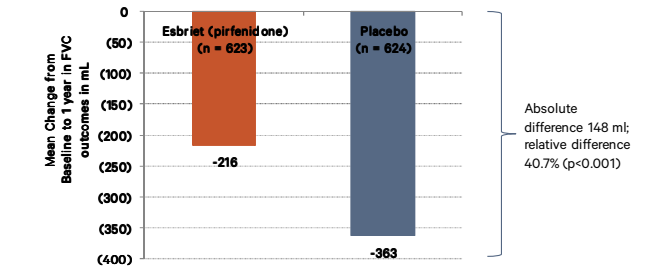
Boehringer recently in-licensed an autotaxin inhibitor in Phase I

On July 19, Boehringer Ingelheim and Bridge Biotherapeutics announced that they entered into a new collaboration and license agreement with the goal of developing Bridge Bio's ATX inhibitor BBT-877 for patients with fibrosing interstitial lung diseases, including IPF. BBT-877 is currently in Phase I clinical studies and is anticipated to enter Phase II testing within the next 12 months. (We note that Boehringer developed Ofev (nintedanib)). **Bridge Bio will receive upfront and near-term payments of €45m and is eligible to receive up to more than €1.1bn in potential payments based on the successful achievement of specified development, regulatory and commercial milestones; Bridge Bio is also eligible for staggered, up to double digit royalties.** To us, this collaboration represents a validation of Galapagos' approach to IPF treatment with an ATX inhibitor given Boehringer's leadership in respiratory disease therapy development broadly, and in IPF in particular.

Bridge Bio recently presented intriguing pre-clinical data for BBT-877 at ATS 2019. Results of comprehensive in vitro and in vivo studies with BBT-877 demonstrate the compound is a very potent and selective ATX inhibitor with a very favorable safety profile, according to Bridge Bio. The data supported further clinical investigation in clinical testing for the treatment of IPF. The poster suggested the Phase I clinical studies are currently ongoing and will be completed by August 2019. See here.

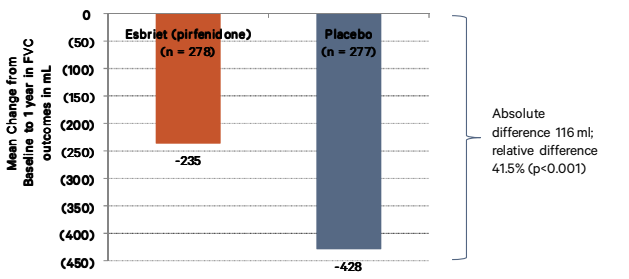
Competitor comparisons: current and emerging IPF therapies

Exhibit 36: Esbriet pooled data points to a near 41% relative improvement in FVC vs. placebo



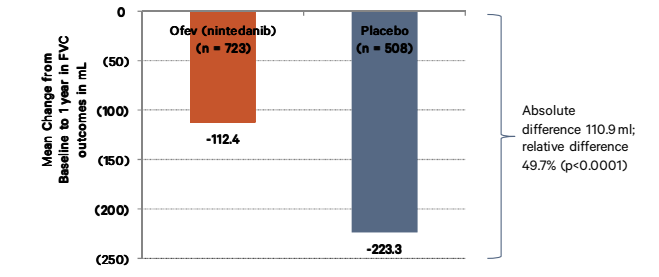
Source: European Respiratory Journal 2015, Berenberg Capital Markets

Exhibit 37: Esbriet confirmatory Phase III study ASCEND also demonstrated an approximate 41% relative improvement in FVC



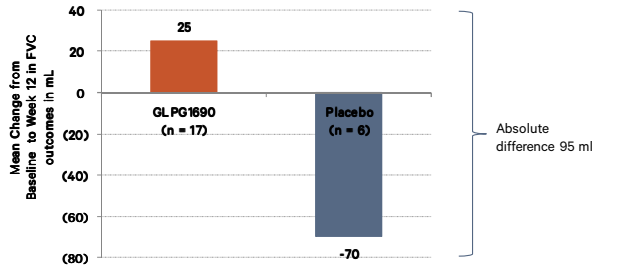
Source: European Respiratory Journal 2015, Berenberg Capital Markets

Exhibit 38: Ofev pooled data demonstrated a near 50% relative improvement in FVC vs. placebo



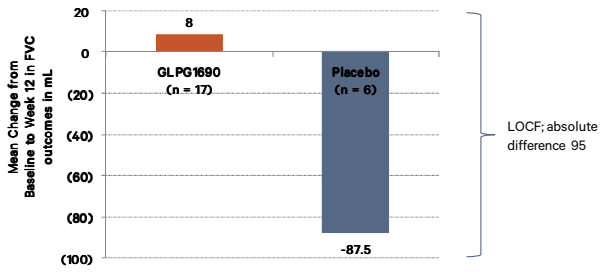
Source: Respiratory Medicine 2016, Berenberg Capital Markets

Exhibit 39: GLPG1690 showed the potential to stabilize FVC as a single agent, which would be unprecedented (FLORA study)



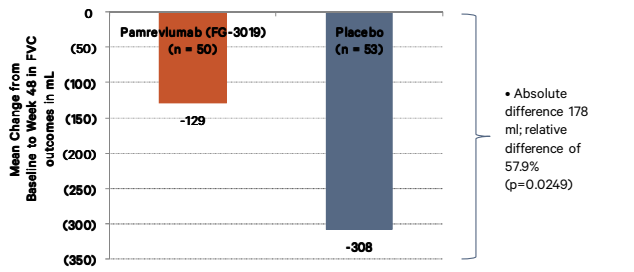
Source: The Lancet, Berenberg Capital Markets

Exhibit 40: GLPG1690 showed the potential to stabilize FVC, which would be unprecedented (FLORA study, Phase II)



Source: The Lancet, Berenberg Capital Markets

Exhibit 41: Pamrev demonstrated an impressive 58% relative improvement in FVC vs. placebo (PRAISE study, Phase II)



Source: The Lancet, Berenberg Capital Markets

Exhibit 42: GLPG1690 and pamrevlumab have shown promise in Phase II studies; separately, although BMS-986020 did not advance, it demonstrated potential for an LPA1 antagonist; as noted earlier in this report, autotaxin is upstream of LPA1

Drug	Company	Trial	Mechanism	Dosing	Route	FVC outcomes	Notable side effects	Source	Next event
GLPG1690	Galapagos / Gilead	ISABELA 1 and 2 (identically designed Phase III trials) (n=750 in each study)	Autotaxin inhibitor	• 600 mg and 200 mg qd	Oral	TBA	TBA	Clinicaltrials.gov	Interim analysis timing update: Q419; primary completion listed as December 2021
GLPG1690	Galapagos / Gilead	FLORA (Phase II) (n=23)	Autotaxin inhibitor	• 600 mg qd	Oral	• Mean change from baseline at Week 12 of +25 ml in GLPG1690 group vs. -70 ml in placebo group • Mean change from baseline at Week 12 (LOCF) of +8 ml in GLPG1690 group vs. -87.5 ml in placebo group			
Pamrevlumab (FG-3019)	FibroGen	Phase III (n=565)	CTGF inhibitor	• 30mg/kg every three weeks	IV	TBA	TBA	Clinicaltrials.gov	Primary completion listed as March 2023
Pamrevlumab (FG-3019)	FibroGen	PRAISE (Phase II) (n=103)	CTGF inhibitor	• 30mg/kg every three weeks	IV	• Mean change from baseline at Week 48 of -129 ml in pamrevlumab group and -308 ml in placebo group • Absolute difference 178 ml; relative difference of 57.9% (p=0.0249)		European Respiratory Journal, 2017, and FGEN 10-K, 2018	
BMS-986020	Bristol-Myers Squibb	Phase II	Lysophosphatidic acid receptor (LPA1) antagonist	• 600 mg qd or bid for 26 weeks	Oral	• BMS-986020 600 bid treatment for 26 weeks vs. placebo significantly slowed the rate of FVC decline	• The study was terminated early because of three cases of cholecystitis that were determined to be treatment-related after unblinding	Chest Journal, 2018	

Source: Chest Journal, European Respiratory Journal, company filings, The Lancet, Clinicaltrials.gov, Berenberg Capital Markets

Exhibit 43: IPF drug data: standard of care therapies including pirfenidone and nintedanib

Drug	Company	Trial	Mechanism	Dosing	Route	FVC outcomes	6MWD or death	PFS	Deaths	Notable side effects	Source
Esbriet (pirfenidone)	Genentech / Roche	Pooled analysis of ASCEND (016), CAPACITY (004), and CAPACITY (006) Phase III trials (n=1247)	Has not been established	• Days 1 through 7: one capsule, tid with meals • Days 8 through 14: two capsules, tid with meals • Days 15 onward: three capsules, tid with meals	Oral	• Mean change from baseline to 1 year -216 ml in pirfenidone group and -363 ml in placebo group • Absolute difference 148 ml; relative difference 40.7% (p<0.001)	• 153 (24.8%) pirfenidone group experienced a ≥ 50m decline in 6MWD or death compared with 214 (34.8%) placebo group (p<0.001)	• Pirfenidone, as compared with placebo, reduced the relative risk of death or disease progression by 38% (p<0.001)	• 22 (3.5%) from any cause; 7 (1.1%) related to IPF, vs. placebo rates of 42 (6.7%) and 22 (3.5%), respectively	• Elevated liver enzymes; photosensitivity and rash; gastrointesinal disorders, notably nausea (35.5% in pirfenidone group vs. 15.1% in placebo group), diarrhea (24.6% vs. 18.8), and rash (29.2% vs. 9%)	European Respiratory Journal, 2015, FDA label
Esbriet (pirfenidone)	Genentech / Roche	ASCEND (n=555) (confirmatory Phase III study)	Has not been established		Oral	• Mean decline from baseline to 1 year was -235 ml in pirfenidone group and -428 ml in placebo group • Absolute difference 116 ml; relative difference 41.5% (p<0.001)	• 72 (25.9%) pirfenidone group experienced a ≥ 50m decline in 6MWD or death compared with 99 (35.7%) placebo group (p<0.001)	• Pirfenidone, as compared with placebo, reduced the relative risk of death or disease progression by 43% (p<0.001)			NEJM, 2014
Drug	**Company**	**Trial**	**Mechanism**	**Dosing**	**Route**	**FVC outcomes**	**Time to first exacerbation**	**SGRQ total score**	**Deaths**	**Notable side effects**	**Source**
Ofev (nintedanib)	Boehringer Ingelheim	Pooled analysis from TOMORROW (Phase II) and INPULSIS (Phase III) programs (n=1231)	Inhibits multiple receptor tyrosine kinases (RTKs) and non-receptor tyrosine kinases (nRTKs)	• 150 mg bid approximately 12 hours apart taken with food	Oral	• Mean change from baseline to 1 year -112.4 ml in nintedanib group and -223.3 ml in placebo group • Absolute difference 110.9 ml ; relative difference 49.7% (p<0.0001)	• The proportion of patients with ≥ 1 acute exacerbation was 4.6% in the nintedanib group and 8.7% in the placebo group (p=0.0047)	• 2.92 in the nintedanib group and 4.97 in the placebo group • Absolute difference of -2.05 in favor of nintedanib (p=0.0095)	• A 30% reduction in the risk of all-cause mortality was observed with nintedanib vs. placebo over 52 weeks (p = 0.0954) • Proportion of patients who died during on-treatment period of 3.5% in nintedanib group and 6.7% in placebo group (p=0.0274)	• Elevated liver enzymes; embryofetal toxicity; arterial thromboembolic events; bleeding; and gastrointestinal disroders, notably diarrhea (61.5% of patients in the nintedanib group vs. 17.9% placebo group) and nausea (24.3% vs. 7.1)	Respiratory Medicine 2016, FDA label

Source: Berenberg Capital Markets

Exhibit 44: Although the primarily completions are 15 months apart, we think ISABELA could generate top-line data well ahead of ZEPHYRUS given the enthusiasm for GLPG1690, which we discuss in further detail in the next sections of this report

	ZEPHYRUS (FibroGen)	ISABELA (Galapagos/Gilead)
Study type	Interventional (clinical trial)	Interventional (clinical trial)
Estimated enrollment	**565 participants**	**1,500 participants**
Allocation	Randomized	Randomized
Intervention model	Parallel assignment	Parallel assignment
Official title	A Phase III, randomized, double-blind, placebo-controlled efficacy and safety study of pamrevlumab in subjects with idiopathic pulmonary fibrosis (IPF)	A Phase III, randomized, double-blind, parallel-group, placebo-controlled multicenter study to evaluate the efficacy and safety of two doses of GLPG1690 in addition to local standard of care for minimum 52 weeks in subjects with idiopathic pulmonary fibrosis
Actual start date	June 27, 2019	November 28, 2018
Estimated primary completion date	**March 2023**	**December 2021**
	Arm:	Arm:
	1. Pamrevlumab 30 mg/kg by intravenous infusion every 3 weeks for a total of 17 infusions over 48 weeks	1. GLPG1690 200 mg will be administered as film-coated tablets for oral use once daily
	2. Placebo	2. GLPG1690 600 mg will be administered as film-coated tablets for oral use once daily
		3. Placebo
Primary outcome measure	**Change in FVC (L) [Time Frame: Baseline to Week 52]**	**Rate of decline of forced vital capacity (FVC) in mL. [Time Frame: From baseline through week 52]**
Secondary outcome measures	1. Change in FVC percent predicted (FVCpp)	1. Disease progression defined as the composite endpoint of first occurrence of ≥10% absolute decline in percent predicted forced vital capacity (%FVC) or all-cause mortality. [Time Frame: At week 52]
	2. Subjects with FVCpp decline of 10% or more or death during study	2. Time to first respiratory-related hospitalization until the end of the study [Time Frame: From screening through study completion, a minimum of 52 weeks]
	3. Change in St. George's Respiratory Questionnaire (SGRQ) score	3. Change from baseline in the St. George's Respiratory Questionnaire (SGRQ) total score. [Time Frame: At week 52]
	4. Composite clinical outcomes including the following: respiratory hospitalization + death + acute IPF exacerbations + FVCpp decline ≥10%	
	5. Change in QLF volume	
	6. Change in University of California San Diego - Shortness of Breath Questionnaire (UCSD-SOBQ)	
	7. Mortality rate	
	8. Acute IPF exacerbations	

Source: Clinicaltrials.gov, Berenberg Capital Markets

Exhibit 45: ZEPHYRUS vs. ISABELA pivotal Phase III studies in IPF: Inclusion criteria notable differences

ZEPHYRUS (FibroGen)	ISABELA (Galapagos/Gilead)
1. Age 40 to 85 years, inclusive, at screening initiation.	**1. Male or female subject aged ≥40 years on the day of signing the Informed Consent Form (ICF).**
2. Diagnosis of IPF as defined by ATS/ERS/JRS/ALAT guidelines (Raghu 2018).	2. A diagnosis of IPF within 5 years prior to the screening visit, as per applicable American Thoracic Society (ATS)/European Respiratory Society (ERS)/Japanese Respiratory Society (JRS)/Latin American Thoracic Association (ALAT) guidelines at the time of diagnosis.
3. History of IPF diagnosis within the past 5 years with onset defined as the date of the first recorded diagnosis of IPF by HRCT and/or SLB in the medical history.	3. Chest high-resolution computed tomography (HRCT) historically performed within 12 months prior to the screening visit and according to the minimum requirements for IPF diagnosis by central review based on subject's HRCT only (if no lung biopsy (LB) available), or based on both HRCT and LB (with application of the different criteria in either situation). If an evaluable HRCT <12 months prior to screening is not available, an HRCT can be performed at screening to determine eligibility, according to the same requirements as the historical HRCT.
4. Interstitial pulmonary fibrosis defined by HRCT scan at Screening, with evidence of ≥10% to <50% parenchymal fibrosis (reticulation) and <25% honeycombing, within the whole lung, as determined by the HRCT central reader.	4. The extent of fibrotic changes is greater than the extent of emphysema on the most recent HRCT scan (investigator-determined)
5. FVCpp value ≥50% and ≤90% at Screening. **6. Diffusing capacity of the lungs for carbon monoxide (DLCO) percent of predicted and corrected by Hb value ≥30% and ≤90% at Screening.**	**5. Meeting all of the following criteria during the screening period: FVC ≥45% predicted of normal, Forced expiratory volume in 1 second (FEV1)/FVC ≥0.7, diffusing capacity of the lung for carbon monoxide (DLCO) corrected for Hb ≥30% predicted of normal**
7. Both FVC and DLCO testing must be representative of the IPF underlying disease.	6. Estimated minimum life expectancy of at least 30 months for non IPF related disease in the opinion of the investigator.
8. Not currently receiving treatment for IPF with approved or unapproved therapy.	**7. Subjects receiving local standard of care for the treatment of IPF, defined as either pirfenidone or nintedanib at a stable dose for at least two months before screening, and during screening; or neither pirfenidone or nintedanib (for any reason). A stable dose is defined as the highest dose tolerated by the subject during those two months.**
9. Male subjects with partners of childbearing potential and female subjects of childbearing potential (including those <1 year postmenopausal) must use double barrier contraception methods during the conduct of the study, and for 3 months after the last dose of study drug.	8. Male subjects and female subjects of childbearing potential agree to use highly effective contraception/preventive exposure measures from the time of first dose of investigational medicinal product (IMP) (for the male subject) or the signing of the ICF (for the female subject), during the study, and until 90 days (male) or 30 days (female) after the last dose of IMP.
10. Able to understand and sign a written informed consent form.	9. Able to walk at least 150 meters during the 6-Minute Walk Test (6MWT) at screening Visit 1; without having a contraindication to perform the 6MWT or without a condition putting the subject at risk of falling during the test (investigator's discretion). The use of a cane is allowed, the use of a stroller is not allowed at all for any condition. At Visit 2, for the oxygen titration test, resting oxygen saturation (SpO2) should be ≥88% with maximum 6 L O2/minute; during the walk, SpO2 should be ≥83% with 6 L O2/minute or ≥88%

Source: Clinicaltrials.gov, Berenberg Capital Markets

KOL view: IPF

We recently spoke with a key opinion leader (KOL) at a major medical practice in California who worked on the pirfenidone studies. The KOL is actively involved in research that aims to better understand the pathogenesis of IPF. The following text is our best-efforts notes and **not** a transcript from the conversation.

Berenberg Capital Markets (BCM) question: How many new patients do you treat each month? The KOL's practice sees a fair number, perhaps 15-20 new diagnoses of IPF each month.

BCM question: What is the standard of care (SOC) treatment for these new patients? The vast majority will receive either nintedanib or pirfenidone. Those who are too severe, such as if they are on four, six, or 10 liters of oxygen, for instance, patients who are in really bad shape, it is not useful to treat patients at this stage. Then there are instances, perhaps six or so per year, where patients present with very early IPF; somehow, it was discovered early, and the person is essentially asymptomatic. Approximately 20% of these early stage patients may not want to start SOC medications because of the side effect profile. This is not the KOL's recommendation, but that is what the patients choose to do.

BCM question: What is the breakdown of patients treated with nintedanib vs. pirfenidone? The split is approximately 50/50 at this particular KOL's practice. For nintedanib, weight loss can be a problem in perhaps 10% of patients; for pirfenidone, photosensitivity, rash, and somnolence can be problematic. Most patients are able to work through the side effect profile. They adjust their diet, etc. If a person starts one drug, 85-90% of the time they are able to stick with that drug; 10-15% of the time, the KOL will switch a patient to the other medication.

BCM question: What is the payor view of SOC medications? It is a pretty standard process to get coverage for the SOC treatments. The KOL could only think of one instance where an insurance company denied approval; in that instance, the insurance company contested the KOL's practice that the patient did in fact have IPF.

BCM question: How do you go about diagnosing IPF? The KOL notes that clinicians rely closely on the diagnosis guidelines, which we outlined earlier in this report. If the HRCT is showing a UIP pattern, then this is IPF; this accounts for approximately 50% of the patients. The remaining 40-50% of patients should undergo a surgical biopsy to make a definitive diagnosis; at least at the KOL's center, this is the recommendation. Not everyone can get a biopsy; the patient may be too sick, too old, or have comorbidities. For the patients that do not undergo a biopsy, the KOL will make a provisional diagnosis rather than a definitive diagnosis; provisional high or provisional low IPF. The KOL notes that in Europe, they do not perform biopsies very often; instead, a bronchoscopy and lavage are performed.

BCM question: The literature points to an increase in prevalence of IPF. Is that accurate and if so, why? The KOL confirms that the prevalence of IPF is increasing. Some of the reasons include increased recognition of the disease and the use of CT scans for at risk patients experiencing shortness of breath. So clinicians are finding it on imaging more often. The other issue is the aging of the population; as the baby boomer generation ages, there are more old people. The majority of people with IPF are 65 years or older; also, around 70% are former smokers.

BCM question: What is the normal rate of lung function decline as defined by FVC? Normal aging, with no lung disease, a person loses around 25-30 ml per year.

BCM question: What about for a patient with IPF? The KOL believes 200-300 ml per year is standard. The KOL noted these rates have been fairly consistent in the placebo rates for studies that evaluated the SOC drugs.

BCM question: What is the improvement shown in real world practice with SOC? Both drugs are approximately equal in terms of cutting the annual rate of decline in FVC about in half for IPF patients.

BCM question: Do the patients who take SOC treatment survive longer? The KOL believes they do. There was a strong trend in the studies. The KOL was involved in the first pirfenidone studies. The KOL notes that the longest a patient surviving on pirfenidone that he can recall was 12 years; some patients were on treatment for 8-9 years. The KOL caveats this by describing the outcomes as anecdotal evidence.

BCM question: For the next generation of IPF drugs, what would be ideal for you to see in the data? The next step is to halt disease progression, as measured by FVC decline, in its tracks, with reasonable tolerability.

BCM question: Broadly, what is your view of GLPG1690 and pamrevlumab? Both drugs under went the usual pathway for drug development for IPF where there's preclinical studies done with fibrosis models that show evidence that these drugs may work; pamrev had a bit of a different preclinical model. After successful Phase I studies, the Phase II studies, in small numbers of patients, over a relatively short treatment interval, showed that both drugs favored people on the drug. The KOL notes that while the bleomycin model is not great, these drugs were studied in humans and both showed a favorable trend, so it makes sense to go to the next step to larger Phase III studies.

BCM question: What is your view of the FLORA data? The KOL believes the trend shown for GLPG1690 in FLORA was favorable; the drug halted the decline in FVC, with the caveat that this study had a very small number of patients. That noted, the KOL believes Galapagos made the right decision based on this data to move forward to a larger Phase III program.

BCM question: What is your view of the PRAISE data? The KOL notes the slightly larger number of patients makes this data slightly more reliable as compared to FLORA, however, at the end of the day, PRAISE also had a small number. The KOL notes that it would not take a lot to sway the p value in one direction or the other; the data was not overwhelmingly positive to the KOL. That noted, the KOL believes FibroGen made the right decision based on this data to move forward to a larger Phase III program.

BCM question: A key difference in the ZEPHYRUS program and ISABELA program is the enrollment of patients on SOC in the ISABELA program vs. a true placebo in the ZEPHYRUS program. What is your view on this? The KOL believes the practicality enrolling a placebo arm of a trial for a terminal disease when drugs are available could be difficult, so the design of ISABELA makes sense to the KOL. For instance, it is more practical to allow patients to continue on SOC; this may make enrollment of ISABELA go smoother as a result, according to the KOL. The other patient group they will want to capture are those who are intolerant to either pirfenidone or nintedanib. Historically, the published literature suggests 40% of IPF patients are unable to tolerate one or the other medications; to the KOL, this number is too high. In the KOL's experience, no more than 10% cannot tolerate SOC medications; however, the point is, if it is in fact as high as 40%, and those patients are captured, then those patients would then have access to a drug that may work. Then there is the placebo arm, where background therapy is also permitted. If everyone is on background therapy, then it is uniform; that is not going to be the case with GLPG1690; the KOL was not sure if specific percentages of patients in each arm would be on background therapy.

Regarding ZEPHYRUS, the KOL sees some risk to enrollment. For instance, if the 40% published number cited above is the right number, then the trial can certainly be enrolled; if it is too high, the KOL sees some risk that FibroGen will be able to find enough patients willing to enroll in the study. On the other hand, the KOL notes that with ZEPHYRUS, we will have a true placebo arm; if pamrev works very well, let's say it cuts progression by 75% or stops disease progression entirely, pamrev would immediately leap to SOC. Thus, while the KOL sees some risk, the KOL believes the intention with ZEPHYRUS is to establish pamrev as a best-across-class drug for IPF.

[Note: At ATS 2019, an abstract poster presented the rates of adherence and persistence of antifibrotic therapies in the U.S. Medicare population; the data suggested that around 75% of pirfenidone and 71% of nintedanib patients continued on their therapy during the study period (January 2010 through December 2015); around 20% of pirfenidone and 26% of nintedanib patients discontinued treatment; approximately 5% of pirfenidone and 3% of nintedanib patients switched drugs.]

BCM question: Regarding the inclusion criteria, does the age difference between ISABELA and ZEPHYRUS matter? The KOL does not think this matters. Once someone is over age 85, it is hard to find someone who can enroll.

BCM question: Regarding the inclusion criteria, do the differences in definitions for FVCpp and DLCO matter? The KOL does not think this matters. IPF patients lose 200-300 ml per year, no matter what stage of the disease, according to the KOL, so there is no need for an upper limit. There are historical reasons for the cut-offs; without them we would not

know if a drug worked late and not early or early and not later on; keep in mind that we learn more about IPF each year and some of these studies were designed when we knew less.

BCM question: Which endpoint is most relevant to you? The change in FVC, ml per year. This is a straight up, hard number. The other endpoints will be helpful to see, but it's really all about FVC to this KOL.

BCM question: Based on what you know about GLPG1690 and pamrevlumab, do you think either compound would be preferable to the other or to SOC? The KOL believes that we really need to see the Phase III data to determine which treatment is preferable over the others. From one perspective, the infusion is okay, because it is one and done; this will be individual dependent, context dependent. The KOL adds that GLPG1690's dosing is convenient; one pill a day is easy. For both treatments, if they are effective and tolerable, these are two huge advances, really. That noted, the KOL believes most people would rather take a pill once a day.

KOL view: IPF

We recently spoke with a key opinion leader (KOL) at a major medical practice in California. The KOL's areas interest include: pulmonary and critical care, specifically interstitial lung disease, lung transplantation, COPD, asthma, and pleural disease. The following text is best efforts notes and **not** a transcript from the conversation.

Berenberg Capital Markets (BCM) question: How many new patients do you treat each month? The KOL sees anywhere from 60-100 IPF patients, as well as a lot of other patients with interstitial lung disease.

BCM question: What is the typical loss of lung function, and what is it for IPF patients, in terms of FVC in ml? A normal person has around 4 liters and starts to lose lung function at around age 25, around 25-30 ml per year. For IPF patients, they can lose 100-200 ml per year; some can lose more than 200 ml per year.

BCM question: What is the typical life expectancy of an IPF patient at your practice? In the KOL's experience, IPF patients are very sick people, maybe they last another six months or one year.

BCM question: What is your view of the current IPF treatment paradigm? There is no good treatment, according to the KOL. We have Ofev and Esbriet. For Esbriet, data was generated in Japan and in the U.S. These were not great studies; CAPACITY 1 showed a reduction in loss of FVC, but CAPACITY 2 did not; in CAPACITY 2, the placebo group performed better than expected. They then took all that data and looked at what patients had the best effect with Esbriet; patients that were more obstructed. So they took out all the obstructed patients, then took the patients with lower DLCOs, then followed these patients for one year, rather than 18 months. The data for Ofev is similar to Esbriet. To the KOL, the honeymoon period is over for these drugs, for many reasons. Theoretically they do slow disease progression, but they do not decrease shortness of breath or improve other symptoms for patients, or really improve the quality of life for patients.

BCM question: Do you have a preference between Ofev and Esbriet? The KOL is using more Ofev rather than Esbriet, mainly due to more favorable tolerability.

BCM question: Are there particular side effects that make either Ofev or Esbriet problematic? Everything is manageable now, according to the KOL. Ofev's biggest problem is diarrhea; Ofev causes a large number of instances of diarrhea in the first three months. Everyone gets diarrhea with a higher dose; the key is to start slow, once a day, 150 mg a day, then go to twice a day. So realistically the side effects are not bad. Also, if a patient takes Ofev on an empty stomach, they will receive a high dose, very quick, which can lead to a nauseated feeling. The same issues can happen with Esbriet, and then there's also some photosensivity that can lead to a rash; key is to wear sunscreen. Liver dysfunction can also be problematic in around 4-5% of patients.

BCM question: do you have patients who switch between the two treatments? The KOL has seen this, but not often; perhaps a few percent of patients switch.

BCM question: What is your view on BG00011 (formerly STX-100) and SPIRIT? Biogen uses an anti-integrin to block the TGF-β pathway; however, the Phase II study (SPIRIT) was stopped prematurely; Biogen has not indicated why the study was stopped. The KOL cited

the early mouse models from around 2000 that showed promise in the TGF-β pathway, noting that there was a reduction of fibrosis; however, the KOL also noted there was an increase in inflammation. Based on this, the KOL postulates that with Biogen enrolling too many relatively younger patients, in their 40s and 50s, these patients may have had too much inflammation, and this may have negatively affected the trial. The KOL adds that the TGF-β hypothesis will take a big hit if BG00011 goes down.

[Note: Galapagos confirmed that the Biogen study was halted and that this is one of the reasons ISABELA is enrolling so much faster than expected.]

BCM question: What is your view of the ATX MOA? This came about off the LPA pathway, a very pulmonary specific pathway; it seemed to reduce a bit of bleomycin-induced fibrosis in mice. The trial to add some validation to this approach was the BMS-986020 Phase II trial where they showed a positive result, statistically significant data (though clinically irrelevant to the KOL). The problem was in the liver function abnormalities, especially for patients on statins. The KOL adds that this looked like a pathway that showed potential for efficacy at least as good as what was out there. ATX is more proximal, but does not have the liver function abnormalities, according to the KOL.

BCM question: What is your view of GLPG1690 data generated to date, including FLORA? The data appears promising. The KOL did not see anything bad with the drug. The KOL notes that if you look at Esbriet or Ofev, it is around 13 weeks where the curves separate; this is similar with the data that was shown with GLPG1690 in FLORA.

BCM question: What is your view of the ISABELA program? The KOL believes Galapagos is smart for taking all comers in the trial. Also, the nice thing is that it's an oral medicine, once a day, so it will be easy for Galapagos to accrue patients. In a way that this study is being conducted, as an add-on therapy, we should see the separation from placebo around 13 weeks, though the key with ISABELA is this: will we see true stability with the combination? If you look at the combination of Ofev and Esbriet, it appears to demonstrate true stability at 12 weeks, so there is some evidence that combining IPF drugs could be helpful. **The KOL emphasized that his patients are asking for GLPG1690.**

BCM question: What are your thoughts on the secondary endpoints? The secondary endpoints will be helpful; definitely want to prevent the 10% drop in FVCpp; looking at exacerbations, only 5-10% have this; regarding St. George's Respiratory Questionnaire, a difference over 10 points is necessary for this to be clinically meaningful in the KOL's view.

BCM question: What is your view of pamrevlumab? CTGF has been around a lot longer. A lot more studies with the knockouts and the effects on fibroblasts. The problem here: Difficult to create the antibody. The biggest problem is that they had difficulty with the production of the antibody. Have to give high doses to get neutralization. Pretty tolerable drug.

BCM question: Is the IV administration going to be a major hurdle for pamrev? The IV is a hurdle; even when you have these patients in clinical trials, when they visit, they have to sit, the clinician has to check them before, and after; it's a huge hurdle. The KOL had heard that pamrev may be reformulated into a subcutaneous formulation; this would be more favorable to the KOL.

BCM question: Is it ethical to enroll IPF patients on a placebo? The KOL views it as being ethical from the standpoint that these drugs are not that effective. As a result, FibroGen will have much cleaner data; a better chance of hitting that p value as a single agent, so the KOL does view FibroGen as being smart in that regard.

BCM question: Is combination therapy the future for the IPF treatment paradigm? The KOL agrees, and referenced the INJOURNEY data cited earlier in this report.

BCM question: If you had to compare the level of interest of patients in GLPG1690 vs. pamrev, how would you characterize it? The KOL indicates that approximately 25% of patients are interested in pamrev and 75% are interested in GLPG1690.

Our view: GLPG1690 could become part of SOC treatment for IPF

The ISABELA trial design appears to be very savvy, taking into account the evolving treatment paradigm and realities of treating a chronic condition. GLPG1690 showed separation from placebo in FLORA at about the time we would expect separation, though in a small patient number. At the same time, INJOURNEY demonstrated that combination

therapy in IPF has potential to improve efficacy without hindering tolerability. **Taken together, the data generated to date, the favorable dosing and administration, the high enthusiasm around GLPG1690 among clinicians and patients, Boehringer's apparent endorsement of the ATX approach, and the potential elimination of a key competitor product in BG00011 give us confidence in raising our peak un-risk adjusted sales estimate to $2bn from $1.5bn.** Request our model for further details.

Filgotinib could generate peak sales of $5bn

U.S. approval in RA is possible earlier in 2020 than we expected

On July 2, Gilead announced that at a recent pre-NDA meeting with the FDA, Gilead provided an update regarding filgotinib. Gilead discussed the FINCH studies and the ongoing MANTA testicular toxicity study assessing semen parameters with filgotinib treatment in men with moderately to severely active ulcerative colitis or Crohn's disease, which together comprise inflammatory bowel disease (IBD). As a result of this discussion, a path forward has been established to submit the NDA for filgotinib as a treatment for rheumatoid arthritis in 2019. We clarified with Galapagos that the FDA had access to the MANTA data generated to date and Galapagos confirmed this, and also that the agency appears to be being more flexible with the filing of an NDA ahead of the full MANTA readout.

This is terrific news because Gilead has a priority review voucher, and if Gilead chooses to use the voucher, the implication is that filgotinib could be approved by mid-2020 in the U.S. We think the Phase III misses in the STELLAR program (NASH) could make it more likely Gilead pivots to filgotinib in RA. However, **for now, we are modeling a launch in the U.S., if approved, in Q420.** See here for more information regarding priority review vouchers.

EU approval in RA around mid-2020 matches our expectations

August 15, Gilead and Galapagos announced that the MAA for filgotinib in RA has been validated and is under evaluation by the European Medicines Agency (EMA). The MAA is supported by the 24-week data from the FINCH program. The filgotinib filing will be reviewed by the EMA under the centralized licensing procedure for all 28 member states of the European Union, as well as Norway, Iceland, and Liechtenstein. **The news is consistent with our modeling assumptions that assume a launch in RA in the EU, if approved, by mid-2020.**

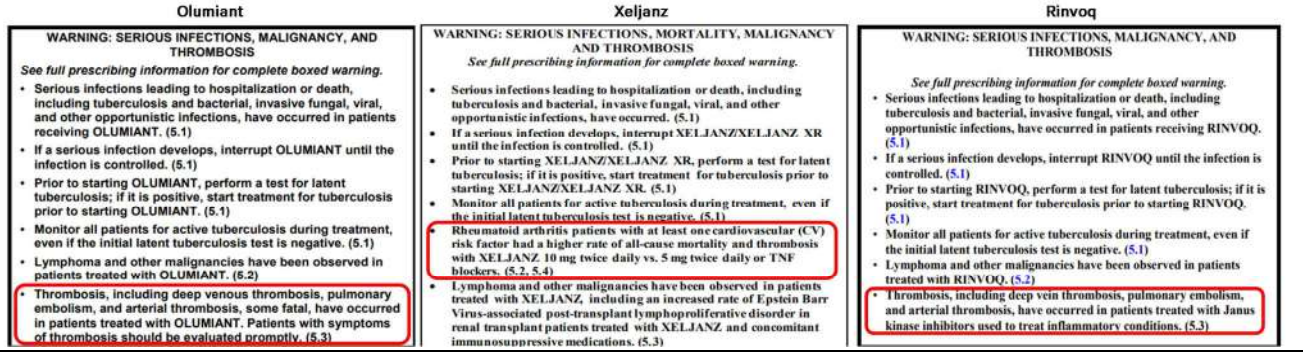
Rinvoq label provides filgotinib an opening to differentiate

On August 16, the FDA approved AbbVie's Rinvoq (upadactinib) 15 mg once-daily for the treatment of adults with moderately to severely active RA who have had an inadequate response or intolerance to methotrexate (MTX-IR). Rinvoq launched in the U.S. in late August. The FDA approval of Rinvoq was supported by data from the SELECT program, one of the largest registrational Phase III programs in RA according to AbbVie with approximately 4,400 patients evaluated across all treatment arms in five studies. Rinvoq is not indicated for methotrexate-naïve patients.

The verbiage regarding thrombosis in the black box warning is broad: can filgo avoid the same fate in the U.S.? The Street therefore believes this will be a class effect for all JAK inhibitors. The key question then is if the FDA will view filgotinib's cleaner safety profile in context to other JAK inhibitors or stick to the broad verbiage used in the black box on the Rinvoq label, which appears very similar to the black box on the Xeljanz label. (See Exhibit 46).

AbbVie did not submit the high dose for upa, and Xeljanz has a thrombosis warning for its high dose, which is only approved for use in UC: can filgo get its high dose approved in RA in the U.S.? Broadly, our discussions with key opinion leaders (KOLs) point to a higher dose JAK being approved for RA as a potentially very significant differentiator. We think filgo's data generated to date supports approval of the high dose (200 mg) in addition to the low dose (100 mg) for RA by the FDA. In the sections that follow, we provide background on the Phase II and III programs for filgotinib in various inflammation indications.

Exhibit 46: Rinvoq's broad language regarding thrombosis has led most investors to believe the FDA views this as a class effect; the expectation is that filgo will have this language too; if it does not, this could be a potentially positive catalyst for GLPG/GILD



Olumiant

WARNING: SERIOUS INFECTIONS, MALIGNANCY, AND THROMBOSIS

See full prescribing information for complete boxed warning.

- Serious infections leading to hospitalization or death, including tuberculosis and bacterial, invasive fungal, viral, and other opportunistic infections, have occurred in patients receiving OLUMIANT. (5.1)
- If a serious infection develops, interrupt OLUMIANT until the infection is controlled. (5.1)
- Prior to starting OLUMIANT, perform a test for latent tuberculosis; if it is positive, start treatment for tuberculosis prior to starting OLUMIANT. (5.1)
- Monitor all patients for active tuberculosis during treatment, even if the initial latent tuberculosis test is negative. (5.1)
- Lymphoma and other malignancies have been observed in patients treated with OLUMIANT. (5.2)
- Thrombosis, including deep venous thrombosis, pulmonary embolism, and arterial thrombosis, some fatal, have occurred in patients treated with OLUMIANT. Patients with symptoms of thrombosis should be evaluated promptly. (5.3)

Xeljanz

WARNING: SERIOUS INFECTIONS, MORTALITY, MALIGNANCY AND THROMBOSIS

See full prescribing information for complete boxed warning.

- Serious infections leading to hospitalization or death, including tuberculosis and bacterial, invasive fungal, viral, and other opportunistic infections, have occurred. (5.1)
- If a serious infection develops, interrupt XELJANZ/XELJANZ XR until the infection is controlled. (5.1)
- Prior to starting XELJANZ/XELJANZ XR, perform a test for latent tuberculosis; if it is positive, start treatment for tuberculosis prior to starting XELJANZ/XELJANZ XR. (5.1)
- Monitor all patients for active tuberculosis during treatment, even if the initial latent tuberculosis test is negative. (5.1)
- Rheumatoid arthritis patients with at least one cardiovascular (CV) risk factor had a higher rate of all-cause mortality and thrombosis with XELJANZ 10 mg twice daily vs. 5 mg twice daily or TNF blockers. (5.2, 5.4)
- Lymphoma and other malignancies have been observed in patients treated with XELJANZ, including an increased rate of Epstein Barr Virus-associated post-transplant lymphoproliferative disorder in renal transplant patients treated with XELJANZ and concomitant immunosuppressive medications. (5.3)

Rinvoq

WARNING: SERIOUS INFECTIONS, MALIGNANCY, AND THROMBOSIS

See full prescribing information for complete boxed warning.

- Serious infections leading to hospitalization or death, including tuberculosis and bacterial, invasive fungal, viral, and other opportunistic infections, have occurred in patients receiving RINVOQ. (5.1)
- If a serious infection develops, interrupt RINVOQ until the infection is controlled. (5.1)
- Prior to starting RINVOQ, perform a test for latent tuberculosis; if it is positive, start treatment for tuberculosis prior to starting RINVOQ. (5.1)
- Monitor all patients for active tuberculosis during treatment, even if the initial latent tuberculosis test is negative. (5.1)
- Lymphoma and other malignancies have been observed in patients treated with RINVOQ. (5.2)
- Thrombosis, including deep vein thrombosis, pulmonary embolism, and arterial thrombosis, have occurred in patients treated with Janus kinase inhibitors used to treat inflammatory conditions. (5.3)

Source: FDA, Berenberg Capital Markets

FINCH program supports solid efficacy, best-in-class safety profile

In August 2016, Gilead initiated the FINCH global Phase III program investigating the efficacy and safety of 100 mg and 200 mg filgotinib once daily, in rheumatoid arthritis (RA) patient populations, ranging from early stage to biologic-experienced patients. **Filgotinib is a selective JAK inhibitor (JAKi) for JAK 1, which we believe contributes to its comparable efficacy to other JAKi, though with a superior safety profile.**

FINCH 1 was a 52-week randomized, double-blind, placebo- and active-controlled study that enrolled 1,759 adult patients with moderately to severely active RA who have an inadequate response to methotrexate (IR-MTX). Eligible patients were randomized (3:3:2:3) to receive filgotinib 200 mg (n=477), filgotinib 100 mg (n=480), adalimumab (AbbVie's Humira) (n=325) or placebo (n=477) in addition to a stable dose of MTX.

The primary endpoint of the study was the proportion of patients who achieve an American College of Rheumatology 20% improvement response (ACR20) at week 12. At week 24, all patients in the placebo arm who did not discontinue study drug were reassigned (1:1) to either filgotinib 100 mg or 200 mg. Developed in 1993, the ACR20 is a composite measure defined as both improvement of 20% in the number of tender and number of swollen joints, and a 20% improvement in three of the following five criteria: patient global assessment, physician global assessment, functional ability measure (most often Health Assessment Questionnaire [HAQ]), visual analog pain scale, and erythrocyte sedimentation rate or C-reactive protein (CRP). ACR50 and ACR70 are the same instruments with improvement levels defined as 50% and 70% respectively versus 20% for ACR20.

Positive top-line data was reported on March 28. The study achieved its primary endpoint for both doses of filgotinib in the proportion of patients achieving an ACR20 compared to placebo at week 12. The proportion of patients achieving ACR50 and ACR70 response was also significantly greater for filgotinib compared with placebo at week 12, for both doses. **With all the caveats of comparing compounds across trials, we note that filgotinib's ACR20/50/70 rates of 70%/36%/19% at 100 mg and 77%/47%/26% at 200 mg compares well to other JAK inhibitors in IR-MTX patients**, including: 1) Xeljanz 5 mg of 61%/34%/12% at three months in the Phase III ORAL-STANDARD trial; 2) Olumiant 4 mg 70%/45%/19% at 12 weeks in the Phase III RA-BEAM trial in IR-MTX patients (and where only the 2 mg dose is approved in the U.S. owing to DVT/PE risk); and 3) upadacitinib 15 mg of 71%/45%/25% at 12 weeks in the Phase III SELECT-COMPARE trial.

In addition, patients receiving filgotinib 100 mg or 200 mg had a statistically significant reduction in the Health Assessment Questionnaire Disability Index (HAQ-DI) at week 12 compared with those receiving placebo. The proportions of patients achieving clinical remission (DAS28(CRP)<2.6) and low disease activity (DAS28(CRP)<3.2) at week 12 were significantly higher for patients in both filgotinib arms compared with placebo. **When comparing low disease activity rates at week 12, filgotinib 200 mg was non-inferior to adalimumab.** Filgotinib 100 mg and 200 mg also significantly inhibited the progression of structural damage at week 24 as assessed by change from baseline in modified total Sharp score (mTSS) compared with placebo.

Exhibit 47: Filgotinib demonstrated significantly higher ACR20/50/70 responses than placebo in patients with IR-MTX

	Week 12							
	Placebo + MTX	Adalimumab 40 mg + MTX	Filgotinib 100 mg + MTX	p-value vs. placebo	p-value vs. ADA	Filgotinib 200 mg + MTX	p-value vs. placebo	p-value vs. ADA
ACR20	49.9%	70.8%	69.8%	<0.001		76.6%	<0.001	
ACR50	19.8%	35.1%	36.3%	<0.001		47.2%	<0.001	
ACR70	6.7%	14.2%	18.5%	<0.001		26.3%	<0.001	
Low disease activity	**23.4%**	**43.4%**	**38.8%**	**<0.001**		**49.7%**	**<0.001**	**<0.001**
Clinical remission	**9.3%**	**23.7%**	**23.8%**	**<0.001**	**<0.01**	**33.9%**	**<0.001**	**<0.01**
Health assessment	-0.42	-0.61	-0.56	<0.001		-0.69	<0.001	
Structural damage (Wk 24)	0.38	0.16	0.17	<0.001		0.13	<0.001	

Note: ADA = adalimumab = AbbVie's Humira; clinical remission p-values vs. ADA not adjusted for multiplicity; low disease activity measured as DAS28(CRP)≤3.2; clinical remission measured as DAS28(CRP)<2.6; Health Assessment Questionnaire Disability Index = HAQ-DI); progression of structural damage at week 24 asses by change from baseline in modified total Sharp score (MTSS) compared with placebo; n= 475 for placebo + MTX, n = 325 for ADA 40 mg + MTX, n = 480 for filgotinib 100 mg + MTX, n = 275 for filgotinib 200 mg + MTX
Source: Company filings, Berenberg Capital Markets

FINCH 2 was a global, 24-week randomized, double-blind, placebo-controlled, Phase III study evaluating filgotinib on a background of conventional synthetic disease-modifying anti-rheumatic drug(s) (csDMARDs) among adult patients with moderately-to-severely active rheumatoid arthritis who had not adequately responded to biologic DMARDs (bDMARDs). In this study, 23.7% of patients had received three or more bDMARDs. Patients were randomized (1:1:1) to receive filgotinib 100 mg, filgotinib 200 mg or placebo. The primary endpoint was the proportion of patients achieving an ACR20 response at week 12. Protocol-defined non-responders at week 14 were allowed to complete the trial under standard of care therapy. Treatment-emergent adverse events are those reported during treatment or within 30 days of the last dose of study drug. **Positive top-line data was reported on September 11.**

Exhibit 48: Filgotinib demonstrated significantly higher ACR20/50/70 responses than placebo in patients with biologics-IR

	Week 12					Week 24				
	Placebo	Filgotinib 100 mg	p-value	Filgotinib 200 mg	p-value	Placebo	Filgotinib 100 mg	p-value	Filgotinib 200 mg	p-value
ACR20	31.1%	57.5%	<0.001	66.0%	<0.001	34.5%	54.9%	<0.001	69.4%	<0.001
ACR50	**14.9%**	**32.0%**	**<0.001**	**42.9%**	**<0.001**	**18.9%**	**35.3%**	**<0.01**	**45.6%**	**<0.001**
ACR70	**6.8%**	**14.4%**	**<0.05**	**21.8%**	**<0.001**	**8.1%**	**20.3%**	**<0.01**	**32.0%**	**<0.001**
Low disease activity	15.5%	37.3%	<0.001	40.8%	<0.001	20.9%	37.9%	<0.01	48.3%	<0.001
Clinical remission	8.1%	25.5%	<0.001	22.4%	<0.001	12.2%	26.1%	<0.01	30.6%	<0.001

Note: low disease activity measured as DAS28(CRP)≤3.2; clinical remission measured as DAS28(CRP)<2.6; Health Assessment Questionnaire Disability Index = HAQ-DI); n= 148 for placebo, n = 153 for filgotinib 100 mg, n = 147 for filgotinib 200 mg
Source: Company filings, Berenberg Capital Markets

We present the top-line data for filgotinib compared to other JAK inhibitors in similar studies in Exhibit 49; with the usual caveats when comparing studies like this that are not directly comparable, filgotinib's placebo-adjusted efficacy on ACR20/50/70 on looks competitive to us.

Exhibit 49: Filgotinib's placebo-adjusted efficacy data compares well to other JAKi

Drug	Company	Trial	Mechanism	Dosing	Route	12 weeks ACR20	ACR50	ACR70
Filgotinib	Galapagos/ Gilead	Phase III	JAK1 inhibitor	100 mg once daily	Oral	26%	17%	8%
				200 mg once daily	Oral	35%	28%	15%
Upadacitinib	AbbVie	Phase III	JAK1 inhibitor	15 mg once daily	Oral	36%	22%	5%
				30 mg once daily	Oral	28%	24%	17%
Baricitinib	Incyte/Eli Lilly	Phase III	JAK1/2 inhibitor	2 mg once daily	Oral	22%	12%	9%
				4 mg once daily	Oral	28%	20%	10%
Tofacitinib	Pfizer	Pooled Phase II/III	Pan JAK inhibitor	5 mg twice daily	Oral	19%	14%	7%
				10 mg twice daily	Oral	27%	17%	9%

Source: Company filings, BMJ, Berenberg Capital Markets

FINCH 3 was an ongoing 52-week randomized, double-blind and active-controlled study examining filgotinib alone and in combination with MTX, enrolling 1,252 adult patients with moderately to severely active RA who are naïve to MTX. Patients were randomized (2:1:1:2) to receive filgotinib 200 mg plus MTX (n=417), filgotinib 100 mg plus MTX (n=207), filgotinib 200 mg alone (n=210) or MTX (n=418). The primary endpoint is the proportion of patients who achieve an ACR20 response at Week 24.

Positive top-line data was reported on March 28. With all the caveats of comparing compounds across trials, we **note filgotinib 200 mg monotherapy generated ACR20/50/70 rates of 78%/58%/40% that compare well to other JAK inhibitors in MTX-naïve patients**, including: 1) Xeljanz 5 mg of 71%/47%/26% at six months in the Phase III ORAL-START trial; 2) Olumiant 4 mg 77%/60%/42% at 24 weeks in the Phase III RA-BEGIN trial (and where only the 2 mg dose is approved in the U.S. owing to DVT/PE risk); and 3) upadacitinib 15 mg of 79%/60%/44% at 24 weeks in the Phase III SELECT-EARLY trial.

Exhibit 50: Filgotinib demonstrated significantly higher ACR20/50/70 responses than MTX in MTX-naïve patients

	Week 24						
	MTX	Filgotinib 100 mg + MTX	p-value	Filgotinib 200 mg + MTX	p-value	Filgotinib 200 mg monotherapy	p-value
ACR20	71.4%	80.2%	<0.05	81.0%	<0.001	78.1%	
ACR50	45.7%	57.0%	<0.01	61.5%	<0.001	58.1%	<0.01
ACR70	26.0%	40.1%	<0.001	43.8%	<0.001	40.0%	<0.001
Clinical remission	29.1%	42.5%	<0.001	54.1%	<0.001	42.4%	<0.001
Health assessment	-0.79	-0.90	<0.01	-0.94	<0.001	-0.89	<0.05
Structural damage	0.52	0.22	NA	0.20	N/A	(0.04)	<0.01

Note: monotherapy p-values vs. MTX not adjusted for multiplicity; clinical remission measured as DAS28(CRP)<2.6; Health Assessment Questionnaire Disability Index = HAQ-DI); progression of structural damage at Week 24 asses by change from baseline in modified total Sharp score (MTSS) compared with MTX; n= 416 for MTX, n = 207 for filgotinib 100 mg + MTX, n = 416 for filgotinib 200 mg + MTX
Source: Company filings, Berenberg Capital Markets

Important note: the placebo rates generated in FINCH 1 and FINCH 3 were unusually high when compared to prior JAKi studies conducted in RA. According to Galapagos, the enrollment criteria in the FINCH program were comparable to other JAKi studies. One explanation that Galapagos offered was that with the JAKi mechanism better understood by patients, this could have influenced the placebo rates, as patients may have believed they were given filgotinib rather than placebo. Galapagos/Gilead understand the uncertainty this creates and will provide additional information in due course; we expect to learn more regarding the baseline characteristics of patients when additional FINCH data is published.

Safety profile is the key potential differentiator for filgotinib, in our view

Clinical trials to date have shown that filgotinib is well-tolerated, with atherogenic index improvement, absence of anemia, low infection rates and low incidence of deep venous thrombosis (DVT) and pulmonary embolisms (PE). This is important because although JAKi benefits include efficacy comparable to biologics with advantages in administration (oral vs. injectable), they also have well-documented safety issues. For instance, Pfizer's Xeljanz (tofacinitib), a pan-JAK inhibitor, was only approved at the low doses (5 mg twice daily; 11 mg once daily) in 2012 as the FDA decided the modest incremental benefit at the high doses was not enough to offset apparent incremental toxicity. On February 19, Pfizer announced that it would transition RA patients who were on tofacitinib 10 mg twice daily to tofacitinib 5 mg twice daily in the FDA post-marketing requirement study A3921133. The action was taken owing to the notification from the tofacitinib Rheumatology Data Safety Monitoring Board (DSMB) of a safety signal regarding the tofacitinib 10 mg twice daily treatment arm. The FDA issued a safety alert on February 25 that a safety clinical trial found an increased risk of blood clots in the lungs and death when a 10 mg twice daily dose of tofacitinib was used in patients with RA; the FDA noted that this dose is only approved for ulcerative colitis (UC) patients. Similarly, Eli Lilly's Olumiant (baricitinib), a JAK1/JAK2 inhibitor, was at first rejected by FDA in 2017 owing to concern regarding the risk/benefit profile across various doses, specifically the rate of thromboembolic events, diagnosed as DVT and PE, which were reported in five patients who received baricitinib during the controlled period of two of seven completed Phase II or Phase III trials in RA. As noted earlier in this report, AbbVie's Rinvoq has a black box that is comparable to Olumiant and Xeljanz, though with broad language regarding the thrombosis risk.

With the usual caveats to comparing data across studies, and with the understanding that we still need more long-term safety data to make a definitive conclusion regarding filgotinib's safety in RA, the data generated in FINCH 2 appears very favorable compared to AbbVie's upadacitinib in a Phase III study called SELECT-BEYOND.

Exhibit 51: In battle of JAK1i, filgotinib appears to have better safety profile

	Filgotinib				Upadacitinib			
	100 mg qd		200 mg qd		15 mg qd		30 mg qd	
Event type	N	%	N	%	N	%	N	%
SAEs	8	5.2%	6	4.1%	18	7.6%	22	9.2%
Opportunistic infections	0	0.0%	0	0.0%	1	0.4%	3	1.3%
Herpes zoster	2	1.3%	2	1.4%	3	1.3%	7	2.9%
DVT/PE	0	0.0%	0	0.0%	3	1.3%	1	0.4%
MACE	1	0.7%	0	0.0%	1	0.4%	1	0.4%
Malignancy excl NMSC	0	0.0%	0	0.0%	2	0.8%	2	0.8%
Deaths	0	0.0%	0	0.0%	1	0.4%	1	0.4%

Note: qd = once-daily
Source: Company filings, Berenberg Capital Markets

Galapagos/Gilead provided compelling pooled safety data on March 28. The 24 week safety data from FINCH 1, 2, and 3 were aggregated, including data from 3,452 patients, of which 2,088 received filgotinib. **Only one DVT/PE was reported across 2,088 patients who received filgotinib and, importantly, none at the low dose.**

Exhibit 52: Filgotinib demonstrated significantly higher ACR20/50/70 responses than MTX in MTX-naïve patients

	Placebo/csDMARD		ADA + MTX 40mg EOW		Filgotinib 100 mg + MTX/csDMARD		Filgotinib 200 mg + MTX/csDMARD		Filgotinib 200 mg		Filgotinib total	
	n=1,039		n=325		n=840		n=1,038		n=210		n=2,088	
	N	%	N	%	N	%	N	%	N	%	N	%
Serious infections	10.0	1.0	8.0	2.5	13.0	1.5	13.0	1.3	3.0	1.4	29.0	1.4
Herpes zoster	4.0	0.4	2.0	0.6	5.0	0.6	6.0	0.6	1.0	0.5	12.0	0.6
DVT/PE	3.0	0.3	0.0	0.0	0.0	0.0	1.0	0.1	0.0	0.0	1.0	<0.1
Death	2.0	0.2	0.0	0.0	1.0	0.1	3.0	0.3	0.0	0.0	4.0	0.2
Malignancy (ex-NMSC)	4.0	0.4	1.0	0.3	1.0	0.1	0.0	0.0	0.0	0.0	1.0	<0.1
MACE	5.0	0.5	1.0	0.3	2.0	0.2	2.0	0.2	1.0	0.5	5.0	0.2

Note: MTX = methotrexate; EOW = every other week; csDMARD = conventional synthetic disease-modifying antirheumatic drug; DVT = deep venous thrombosis; PE = pulmonary embolism; NMSC = non-melanoma skin cancer; MACE = major adverse cardiac events; DVT/PE data excludes one retinal vein occlusion; all events except for deaths were treatment-emergent events
Source: Company filings, Berenberg Capital Markets

Robust DARWIN program provides additional support for filings

Galapagos reported positive Phase IIb data in 2015 (DARWIN 1, 2) and 2017 (DARWIN 3). In both DARWIN 1 (24 weeks, 594 patients, add-on to methotrexate, MTX) and DARWIN 2 (24 weeks, 283 patients, monotherapy), Phase IIb dose-range finding clinical trials in insufficient MTX responders with moderate-to-severe RA, filgotinib achieved the primary endpoint of ACR20.

In DARWIN 1, overall, there was no statistically relevant difference between the once-daily and twice-daily dosing regimens. Both trials showed a rapid onset of action, as of week one for ACR and DAS28-CRP responses.

In DARWIN 1 (200 mg twice-daily) and in DARWIN 2 (100 mg once-daily) up to 50% of the patients reached low disease activity or remission. The 100 mg and 200 mg once-daily doses achieved similar levels of activity overall.

Long-term activity levels and safety profile provided by DARWIN 3 were also positive. In DARWIN 3, a multi-center, open-label, long-term follow-up safety and efficacy trial of subjects who have completed either DARWIN 1 or DARWIN 2, all subjects started the trial at the same dose level, either at 200 mg once-daily or 100 mg twice-daily (except for males in the U.S. sites of these trials who received a maximum daily dose of 100 mg), depending on the regimen administered during the preceding trial, with DARWIN 1 subjects continuing to use filgotinib in combination with MTX.

Galapagos/Gilead reported findings from DARWIN 3 at 60 and 84 weeks of treatment in the course of 2017; promising activity levels were maintained and favorable findings related to the tolerability profile were reported. For instance, based on "observed case" analysis, 86%, 69%, and 47% of 560 subjects achieved ACR20/50/70, respectively, and 71% (386/543) achieved DAS28-CRP ≤3.2. Compelling long-term (84-week) safety results were presented at ACR 2017.

More recently, on March 28, Galapagos/Gilead provided safety data through 156 weeks or longer that looks very compelling to us, including just two DVT/PE and much lower rates of infections and herpes zoster compared to other JAKi.

Exhibit 53: Filgotinib's long-term safety data compares well to other JAKs and biologics for RA

Event per 100 PYE	Filgotinib 100 and 200 mg	Baricitinib 2 and 4 mg qd	Tofacitinib 5 mg bid	Upadacitinib 6 and 12 mg qd	Tocilizumab 4 and 8 mg/kg	Adalimumab
PYE	2,203	6,637	5,278	725	14,994	23,943
Serious infection	1.2	2.9	2.4	2.3	4.5	4.6
Herpes zoster	1.5	3.2	3.8	3.7	ND	ND
DVT/PE	0.1	0.5	0.2	0.7	ND	ND
Deaths	0.2	0.3	0.6	0.3	0.6	0.8
Source	DARWIN3	ACR2017	ACR2017	ACR2017	ACR2012	Burmester 2011

Note: PYE = patient year experience; DARWIN 3 was the long-term open-label extension portion of the Phase II DARWIN program evaluating filgotinib in RA patients
Source: Company filings, Berenberg Capital Markets

Filgotinib has also generated compelling data in other indications

Inflammatory bowel disease (IBD) – Phase III date expected in 2020

Filgotinib generated very compelling Phase II data in anti-TNF naïve CD patients. The FITZROY Phase II trial evaluated once-daily filgotinib in 174 patients versus placebo in patients with moderate-to-severely active CD and mucosal ulceration. Patients recruited were either anti-TNF naïve or anti-TNF failures. We note that FITZROY was the first trial in CD to require endoscopic confirmation of lesions at entry, and also to include a placebo control on endoscopy.

The trial comprised two parts, each of 10 weeks duration: the first part investigated the safety and efficacy of filgotinib 200 mg once daily versus placebo, while the second part of the trial investigated continued treatment through 20 weeks in an observational exploratory design.

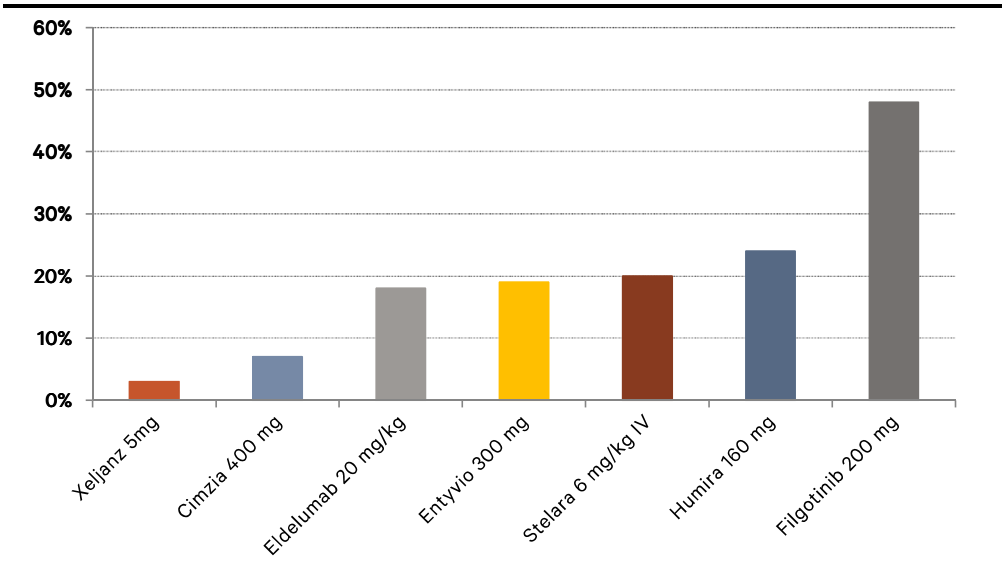
The FITZROY trial achieved the primary endpoint of clinical remission at 10 weeks: the percentage of patients overall achieving a Crohn's Disease Activity Index (CDAI) score lower than 150 was statistically significantly higher in patients treated with filgotinib (47%) versus patients receiving placebo (23%). The share of patients achieving 100 points clinical

response (60%) also was significant versus those receiving placebo (41%). Clinical responses were maintained from week 10 to week 20. Non-responders in the placebo arm from the first ten weeks received filgotinib 100 mg in the second ten weeks and showed improvement in clinical remission during the second part of the trial.

Overall, in the FITZROY trial at 20 weeks of treatment, filgotinib demonstrated a favorable safety profile consistent with the DARWIN trials in RA. An increase in hemoglobin was also observed in FITZROY, without difference between filgotinib and placebo. No clinically significant changes from baseline in neutrophils or liver function tests were observed.

Exhibit 54: Filgotinib performs very well in anti-TNF naïve patients

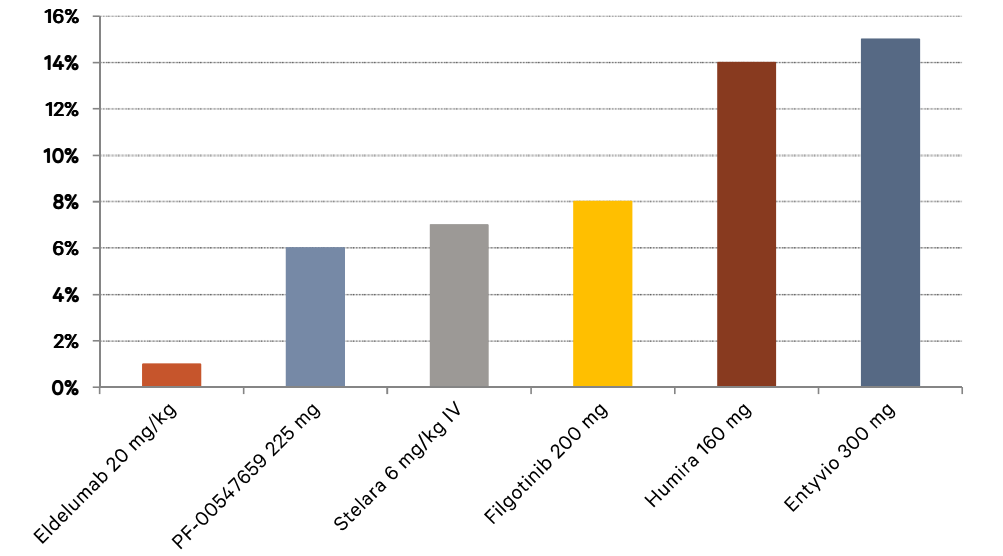
Expressed as % remission, induction study, placebo-adjusted



Source: Company filings, Berenberg Capital Markets

Exhibit 55: Filgotinib's efficacy is comparable to Stelara in patients who failed anti-TNF therapy

Expressed as % remission, induction study, placebo-adjusted



Source: Company filings, Berenberg Capital Markets

Gilead initiated a Phase III trial (DIVERSITY) with filgotinib in CD in November 2016. DIVERSITY will investigate efficacy and safety of 100 mg and 200 mg filgotinib once-daily compared to placebo in patients with moderately to severely active disease including those with prior antibody therapy failure. Gilead will recruit approximately 1,300 patients from the U.S., Europe, Latin America, Canada, and Asia/Pacific regions. Men and women in the

DIVERSITY trial will be randomized to receive placebo, 100 mg or 200 mg filgotinib. In the U.S., males may receive 200 mg if they failed at least one anti-TNF and vedolizumab, a monoclonal anti-integrin antibody sold by Takeda. **Gilead expects to complete recruitment for DIVERSITY in H220.** Refer to details, here.

Gilead initiated the SELECTION Phase IIb/III trial in UC in December 2016. SELECTION investigates efficacy and safety of 100 mg and 200 mg filgotinib once-daily compared to placebo in patients with moderately to severely active disease including those with prior antibody therapy failure. Gilead will recruit approximately 1,300 patients from the U.S., Europe, Latin America, Canada, and Asia/Pacific regions. SELECTION included a futility analysis, serving as the Phase IIb part of this integrated Phase II/III trial. Men and women in SELECTION will be randomized to receive placebo, 100 mg or 200 mg filgotinib. In the U.S., males may receive 200 mg if they failed at least one anti-TNF and vedolizumab. Refer to details, here.

Filgotinib advanced to Phase III in UC last May. On May 30, 2018, Galapagos/Gilead announced that the independent Data Monitoring Committee (DMC) conducted a planned interim futility analysis after 350 patients completed the induction period in the Phase IIb portion of the study. The DMC recommended that the study proceed into Phase III as planned at both the 100 mg and 200 mg once-daily dose level in biologic-experienced and biologic-naïve patients. Galapagos received a $15m payment from Gilead for this progression from Phase II to Phase III in the SELECTION trial. **SELECTION is fully recruited, which implies top-line data should be available around mid-2020.**

Separately, we note that in March 2017, Gilead initiated a Phase II trial in small bowel CD and a Phase II trial in fistulizing CD. These trials are currently recruiting.

Psoriatic arthritis (PsA) – Phase III study start expected H219

Galapagos/Gilead announced positive Phase II data (EQUATOR) in April 2018. EQUATOR was a multi-center, randomized, double-blind, placebo-controlled trial that assessed the safety and efficacy of filgotinib 200 mg once-daily treatment in adult patients with moderately to severely active PsA. The primary goal of EQUATOR was to evaluate the effect of filgotinib compared to placebo on the signs and symptoms of PsA as assessed by the ACR20 at week 16. The trial also explored the effects of filgotinib on the skin manifestations (psoriasis), as well as other domains like fingers (dactylitis), tendon insertions (tendinitis), spine involvement (spondylitis) and nail involvement.

Between March 9 and September 27, 2017, 191 patients in eight European countries were screened and 131 were randomly allocated to treatment (65 to filgotinib 200 mg and 66 to placebo); 60 (92%) patients in the filgotinib group and 64 (97%) patients in the placebo group completed the study; five patients (8%) in the filgotinib group and two patients (3%) in the placebo group discontinued treatment.

Filgotinib met the primary endpoint in EQUATOR; 52 (80%) of 65 patients in the filgotinib group and 22 (33%) of 66 in the placebo group achieved ACR20 at week 16 (treatment difference 47%, p<0.0001). The placebo-adjusted proportion of patients who achieved improvement in ACR20/50/70 was approximately 47%/33%/17%. With all the caveats of cross-trial comparisons, we note that, in a Phase III study (OPAL Broaden, n=422, 12 weeks), Pfizer's Xeljanz, a pan-JAKi, generated placebo-adjusted ACR20/50/70 rates of 17%/18%/12% at the 5 mg dose and 28%/30%/9% at the 10 mg dose.

In terms of safety, 37 (57%) patients who received filgotinib and 39 (59%) patients who received placebo had at least one treatment-emergent adverse event. Six participants had an event that was grade 3 or worse. The most common events were nasopharyngitis and headache, occurring at similar proportions in each group. One serious treatment-emergent adverse event was reported in each group (pneumonia and hip fracture after a fall), one of which (pneumonia) was fatal in the filgotinib group. The full results were published in *The Lancet*.

Ankylosing spondylitis (AS) – Phase III study start expected H120

Galapagos/Gilead announced positive Phase II data (TORTUGA) in September 2018. TORTUGA was a multi-center, randomized, double-blind, placebo-controlled trial to assess the safety and efficacy of filgotinib in adult patients with moderate to severely active AS. The primary goal of TORTUGA was to evaluate the effect of filgotinib compared to placebo on the signs and symptoms of AS, as assessed by the Ankylosing Spondylitis Disease

Activity Score (ASDAS) at week 12. The trial also explored signs and symptoms of AS, physical function, spinal mobility, enthesitis, spinal and sacroiliac joint inflammation, and safety.

Between March 7, 2017, and July 2, 2018, 263 patients in eight European countries were screened and 116 randomly assigned to filgotinib (n=58) or placebo (n=58); 55 (95%) patients in the filgotinib group and 52 (90%) in the placebo group completed the study; three (5%) patients in the filgotinib group and six (10%) in the placebo group discontinued treatment.

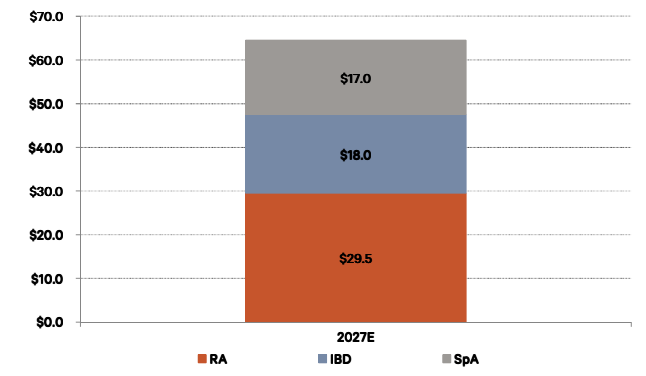
TORTUGA met the primary endpoint; the mean ASDAS change from baseline to week 12 was −1.47 in the filgotinib group and −0.57 in the placebo group (p<0.0001). In addition, approximately 76% of patients who received filgotinib achieved an ASAS20 (Assessment in Ankylosing Spondylitis response, at least 20% improvement), versus 40% of patients who received placebo (p<0.0001).

Treatment-emergent adverse events were reported in 18 patients in each group, the most common being nasopharyngitis (in two patients in the filgotinib group and in four patients in the placebo group). Treatment-emergent adverse events led to permanent treatment discontinuation in two patients, including a case of grade 3 pneumonia in the filgotinib group and of high creatine kinase in the placebo group. No deaths were reported during the study. The full results were published in *The Lancet*.

Bottom line: Filgo's broad label may help it reach $5bn in peak sales

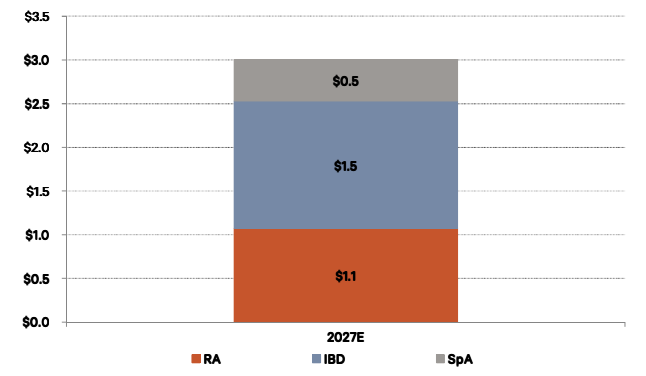
With efficacy competitive to other JAKi, we think the totality of data, including from the FINCH and DARWIN programs, should help Galapagos/Gilead make the commercial case for comparable efficacy with best-in-class safety for filgotinib in the above mentioned indications, among others. To put the $5bn in perspective, if the market for RA, IBD, and SpA reaches nearly $65bn by 2027, our $3bn estimate in these indications would represent just 3% of the market. If filgotinib exclusivity is maintained through the early 2030s, we think $5bn in peak revenues in all inflammation indications is possible. Reach out to us for an Excel version of our model for our complete assumptions for filgotinib.

Exhibit 56: Therapies for inflammation indications RA, IBD, and SpA could reach more than $60bn by 2027E ($m)



Source: Galapagos, Evaluate Pharma, BCM estimates

Exhibit 57: To us, filgotinib market share of 4% in RA, 8% in IBD, and 3% in SpA is very reasonable in 2027E ($m)



Source: Galapagos, Evaluate Pharma, BCM estimates

Valuation and risks

Our discounted cash flow (DCF) and sum-of-the-parts (SOTP) valuation point to an equity value per share of €200 (vs. prior €140), which represents more than 30% potential upside to the current share price; for this reason, we reiterate our Buy rating on Galapagos (GLPG). Changes in our SOTP valuation greater than €1/share are explained in detail below.

Filgotinib. We moderated the value of filgotinib in our model, primarily as now Galapagos will be responsible for 50% of the costs of development, rather than 20% of the costs. With filgotinib being developed in late stage studies in IBD and SpA, this could lead to significant incremental costs compared to what we had previously modeled. On the other hand, with our model at a 70% probability of success in these indications, if filgotinib is eventually approved in these indications, we estimate the value of filgotinib could reach more than €80/share. As it is, we value filgotinib at €62/share (vs. prior €78/share).

GLPG1690. We raised the value of GLPG1690 even as it is now partnered in the U.S. with Gilead primarily as we have upped the probability of success in the program to 70% (from 50%). Given that GLPG1690 had not previously been evaluated in combination with SOC, we considered safety to be a key concern; how would combining these agents impact the side effect profile in a difficult to treat disease in an older patient population? With Gilead having access to blinded safety data, we think this aspect of the story is somewhat derisked, with the caveat that the data was likely based on a relatively small number of patients. If GLPG1690 is eventually approved for treatment of IPF, we estimate the value of GLPG1690 reach €50/share. As it is, we value GLPG1690 at €30/share (vs. prior €22/share).

GLGP1972. We model GLPG1972 as if Gilead chooses to in-license this asset. We also upped the probability of success in the program to 25% (vs. prior 20%) for the same reason as we raised the POS for GLPG1690; Gilead's access to the blinded safety data that gave it confidence to move forward with a robust milestones agreement. We have long believed that GLPG1972, though higher risk, could become Galapagos' most valuable program. If GLPG1972 is eventually approved for treatment of OA knee pain, we estimate the value of GLPG1972 could exceed €70/share; additional indications would be incremental. As it is, we value GLPG1972 at €19/share (vs. prior €15/share).

Platform value. We are now attributing a platform value of €7/share to GLPG shares; we had not previously broken out the platform value separately, though it was implicit within our DCF. We think our assigned platform value could prove to be conservative, particularly if further pipeline success is demonstrated on GLPG1690 and MOR106.

Cash and securities, net. The value of €85/share includes the proceeds from the expanded Gilead collaboration. Gilead was asked on the collaboration expansion conference call following the announcement of the deal how to break down the payments; Gilead did not define the breakdown, and so we do not allocate it to the pipeline. Instead, we moved our DCF forward, to beginning in 2020E, and include the cash from 2019E in our base DCF and SOTP analysis.

Exhibit 58: We raised our price target to €200 (from €140)

€ in millions, unless noted

Sum of the parts valuation	Per share	FCFF DCF valuation	
Filgotinib (Gilead U.S., EU)	€ 62	Terminal Value	14,608
GLPG1690 (Gilead U.S.)	€ 30	PV of Free Cash Flow	4,633
GLPG1972 (Servier EU / Gilead U.S.)	€ 19	PV of Terminal Value	2,669
MOR106 (Novartis, MorphoSys)	€ 8	Implied Enterprise Value	7,302
CF program (AbbVie)	€ 5	Plus: Cash and Securities (Q419)	5,396
Platform value	€ 7	Less: Total Debt (Q419)	0
Cash and Securities, net	€ 85	Implied Value of Equity	12,698
All Other	-€ 16	Diluted Shares Outstanding	63
Implied Value	**€ 200**	**Implied Value per Share**	**€ 200**

Source: Company filings, Berenberg Capital Markets

The key risk to our thesis is disappointing clinical data readouts on important assets such as filgotinib (RA, IBD, and other indications), GLPG1690 (IPF), GLPG1972 (OA knee), and MOR106 (AtD).

Additional risks to our thesis include: 1) drug development risk; 2) competitive risks, including if competitor products in development generate superior clinical data or if competitors conduct commercialization activities better than Galapagos or its partners; 3) government regulatory risk; 4) payer reimbursement risk; 5) pricing risk; 6) capital market risk; and 7) business development risk, among others.

Financials

Profit and loss account

€ in millions, unless otherwise noted

	2013A	2014A	2015A	2016A	2017A	1Q18A	2Q18A	3Q18A	4Q18A	2018A	1Q19A	2Q19A	3Q19E	4Q19E	2019E	2020E	2021E
Total revenues and other income	**96.6**	**90.0**	**60.6**	**151.6**	**155.9**	**44.8**	**57.0**	**103.2**	**112.8**	**317.8**	**40.9**	**67.6**	**3,617.2**	**65.2**	**3,790.9**	**262.1**	**324.3**
% chg		-6.8%	-32.7%	150.3%	2.8%	12.5%	72.0%	209.7%	127.5%	103.9%	-8.7%	18.5%	3404.8%	-42.2%	1092.7%	-93.1%	23.8%
Cost of sales	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	20.0	16.2
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	-18.9%
% of sales	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	7.6%	5.0%
Gross profit	**96.6**	**90.0**	**60.6**	**151.6**	**155.9**	**44.8**	**57.0**	**103.2**	**112.8**	**317.8**	**40.9**	**67.6**	**3,617.2**	**65.2**	**3,790.9**	**242.1**	**308.1**
% chg		-6.8%	-32.7%	150.3%	2.8%	12.5%	72.0%	209.7%	127.5%	103.9%	-8.7%	18.5%	3404.8%	-42.2%	1092.7%	-93.6%	27.3%
% of sales	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	92.4%	95.0%
bps chg		0	0	0	0	0	0	0	0	0	0	0	0	0	0	(760)	260
Research and development expenses	**99.4**	**111.1**	**129.7**	**139.6**	**218.5**	**69.8**	**81.7**	**80.3**	**91.1**	**322.9**	**83.2**	**94.4**	**100.5**	**115.5**	**393.6**	**424.2**	**409.2**
% chg		11.8%	16.7%	7.6%	56.6%	55.3%	70.2%	42.6%	31.5%	47.8%	19.3%	15.5%	25.1%	26.8%	21.9%	7.8%	-3.5%
% of sales	102.9%	123.4%	214.1%	92.1%	140.1%	155.6%	143.2%	77.8%	80.8%	101.6%	203.3%	139.6%	2.8%	177.2%	10.4%	161.9%	126.2%
General and administrative expenses	**12.4**	**13.9**	**19.1**	**21.7**	**24.4**	**6.7**	**8.5**	**9.7**	**10.7**	**35.6**	**9.2**	**13.7**	**14.0**	**14.5**	**51.4**	**60.0**	**75.0**
% chg		12.3%	37.9%	13.7%	12.3%	19.5%	34.4%	66.2%	61.4%	45.9%	37.7%	61.2%	44.0%	35.4%	44.3%	16.7%	25.0%
% of sales	12.8%	15.4%	31.6%	14.3%	15.7%	14.9%	14.9%	9.4%	9.5%	11.2%	22.5%	20.3%	0.4%	22.2%	1.4%	22.9%	23.1%
Sales and marketing expenses	**1.5**	**1.0**	**1.2**	**1.8**	**2.8**	**0.4**	**0.6**	**0.9**	**2.2**	**4.1**	**1.7**	**3.9**	**4.4**	**4.9**	**14.9**	**45.0**	**80.0**
% chg		-32.2%	19.2%	51.0%	57.0%	-25.7%	12.7%	11.1%	146.7%	47.9%	322.8%	543.7%	387.2%	118.3%	258.7%	202.6%	77.8%
% of sales	1.5%	1.1%	2.0%	1.2%	1.8%	0.9%	1.1%	0.9%	2.0%	1.3%	4.3%	5.7%	0.1%	7.5%	0.4%	17.2%	24.7%
Restructuring and integration costs	0.3	0.7	0.0		0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total operating expenses	**113.5**	**126.6**	**150.0**	**163.1**	**245.7**	**76.9**	**90.8**	**90.9**	**104.1**	**362.7**	**94.2**	**112.0**	**118.9**	**134.9**	**459.9**	**529.2**	**564.2**
% chg		11.6%	18.5%	8.7%	50.7%	50.5%	65.5%	44.4%	35.5%	47.6%	22.5%	23.3%	30.7%	29.6%	26.8%	15.1%	6.6%
% of sales	117.5%	140.7%	247.6%	107.6%	157.6%	171.5%	159.2%	88.1%	92.3%	114.1%	230.1%	165.6%	3.3%	206.9%	12.1%	201.9%	173.9%
Operating profit (loss)	**(16.9)**	**(36.6)**	**(89.4)**	**(11.5)**	**(89.8)**	**(32.0)**	**(33.8)**	**12.3**	**8.7**	**(44.8)**	**(53.2)**	**(44.4)**	**3,498.3**	**(69.7)**	**3,331.0**	**(287.1)**	**(256.1)**
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
% of sales	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
bps chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Depreciation and amortization	8.2	4.6	3.4	4.2	4.3	1.2	2.5	1.1	0.3	5.1	2.8	2.9	2.9	2.9	11.4	12.0	13.0
IFRS EBITDA	**(8.8)**	**(32.0)**	**(86.0)**	**(7.3)**	**(85.5)**	**(30.8)**	**(31.3)**	**13.4**	**9.0**	**(39.7)**	**(50.5)**	**(41.5)**	**3,501.2**	**(66.8)**	**3,342.5**	**(275.1)**	**(243.1)**
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
% of sales	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
bps chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Share subscription agreement	0.0	0.0	(30.6)	57.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other financial income	2.2	2.3	2.0	10.0	4.9	1.6	6.5	2.6	7.7	18.3	7.0	(1.3)	1.5	1.5	8.7	8.7	8.7
Other financial expenses	(1.4)	(0.9)	(1.5)	(1.7)	(30.6)	(6.8)	5.6	(0.5)	(1.0)	(2.7)	(2.3)	(1.5)	(1.0)	(1.0)	(5.8)	(5.8)	(5.8)
Total non-operating income (expense)	**0.8**	**1.4**	**(30.2)**	**65.7**	**(25.7)**	**(5.2)**	**12.1**	**2.1**	**6.6**	**15.6**	**4.7**	**(2.8)**	**0.5**	**0.5**	**2.9**	**2.9**	**2.9**
Pretax income (loss)	**(16.1)**	**(35.2)**	**(119.6)**	**54.2**	**(115.5)**	**(37.2)**	**(21.7)**	**14.4**	**15.3**	**(29.2)**	**(48.6)**	**(47.1)**	**3,498.8**	**(69.2)**	**3,333.9**	**(284.2)**	**(253.2)**
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
% of sales	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Taxes	0.7	2.1	(1.2)	0.2	0.2	0.1	0.1	(0.5)	0.4	0.1	0.1	0.1	0.0	0.0	0.1	0.0	0.0
Tax rate	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
IFRS net income (loss)	**(16.8)**	**(37.3)**	**(118.4)**	**54.0**	**(115.7)**	**(37.3)**	**(21.8)**	**14.8**	**15.0**	**(29.3)**	**(48.7)**	**(47.2)**	**3,498.8**	**(69.2)**	**3,333.8**	**(284.2)**	**(253.2)**
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
% of sales	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
IFRS EPS	**€ -0.58**	**€ -1.24**	**€ -3.32**	**€ 1.14**	**€ -2.34**	**€ -0.73**	**€ -0.42**	**€ 0.28**	**€ 0.27**	**€ -0.56**	**€ -0.89**	**€ -0.86**	**€ 56.30**	**€ -1.11**	**€ 56.98**	**€ -4.48**	**€ -3.93**
% chg		NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Shares outstanding	28.8	30.1	35.7	47.3	49.5	51.0	51.3	54.3	54.5	52.1	54.6	54.9	62.1	62.4	58.5	63.4	64.4

Source: Company data, BCM estimates

Balance sheet

€ in millions, unless otherwise noted

	2013A	2014A	2015A	2016A	2017A	1Q18A	2Q18A	3Q18A	4Q18A	2018A	1Q19A	2Q19A	3Q19E	4Q19E	2019E	2020E	2021E
Inventories	0.2	0.3	0.3	0.0	0.0	0.3	0.3	0.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	10.0	20.0
Trade and other receivables	19.2	3.2	3.9	9.7	28.0	8.5	19.1	25.3	18.6	18.6	15.3	42.1	45.1	48.1	48.1	58.1	68.1
Current R&D incentives receivables	10.6	7.4	9.2	10.2	11.8	11.6	14.7	11.7	11.2	11.2	11.6	11.6	11.6	11.6	11.6	11.6	11.6
Cash and cash equivalents	138.2	187.7	340.3	973.2	1,151.2	1,108.2	1,066.8	1,343.7	1,290.8	1,290.8	1,222.9	1,147.9	5,483.4	5,396.4	5,396.4	5,138.4	4,912.3
Current restricted cash	0.0	10.4	6.9	0.0	0.0	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Current financial asset, share sub. agreement	0.0	0.0	8.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other current assets	5.1	4.6	5.5	14.1	6.7	7.2	7.1	7.4	8.2	8.2	9.4	7.0	7.0	7.0	7.0	7.0	7.0
Total current assets	**173.3**	**213.6**	**374.5**	**1,007.2**	**1,197.6**	**1,135.7**	**1,107.9**	**1,390.4**	**1,328.9**	**1,328.9**	**1,259.2**	**1,208.6**	**5,547.1**	**5,463.0**	**5,463.0**	**5,225.1**	**5,019.0**
Goodwill	39.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Intangible assets	7.8	2.0	1.6	1.0	2.5	2.6	1.4	2.1	3.6	3.6	6.5	7.2	7.2	7.2	7.2	7.2	7.2
Property, plant, and equipment	19.5	10.1	13.8	15.0	16.7	17.0	17.9	18.1	23.1	23.1	49.5	51.2	156.6	155.4	155.4	154.9	156.6
Deferred tax assets	4.6	0.3	1.7	2.0	2.0	2.0	2.0	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Non-current R&D incentives receivables	39.3	43.9	49.4	54.2	64.0	69.3	71.6	68.8	73.4	73.4	76.0	82.6	82.6	82.6	82.6	82.6	82.6
Non-current restricted cash	3.3	0.3	1.0	0.0	0.0	1.2	1.2	1.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other non-current assets	0.2	0.2	0.6	4.0	3.5	2.2	2.5	2.5	7.9	7.9	6.4	5.7	5.7	5.7	5.7	5.7	5.7
Total assets	**287.4**	**270.5**	**442.5**	**1,083.3**	**1,286.3**	**1,229.9**	**1,204.3**	**1,485.6**	**1,439.5**	**1,439.5**	**1,400.2**	**1,357.8**	**5,801.7**	**5,716.5**	**5,716.5**	**5,478.0**	**5,273.6**
Provisions	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Lease liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.6	0.0	0.0	0.0	0.0	0.0	0.0
Finance lease liabilities	0.2	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	5.1	5.1	5.1	5.1	5.1	5.1
Trade and other payables	29.4	30.0	29.5	31.9	48.3	55.7	69.1	80.7	68.9	68.9	69.9	86.2	86.2	86.2	86.2	96.2	106.2
Current tax payable	0.1	2.6	2.6	1.0	0.9	0.9	0.9	0.9	1.2	1.2	1.2	1.0	1.0	1.0	1.0	1.0	1.0
Accrued charges	3.9	0.6	0.5	0.0	0.0	0.9	0.9	0.9	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred income	79.0	27.0	39.8	70.8	122.5	166.2	175.7	186.7	149.8	149.8	123.8	96.3	73.3	49.3	49.3	49.3	49.3
Total current liabilities	**112.6**	**60.4**	**72.4**	**103.8**	**171.7**	**223.6**	**246.6**	**269.1**	**219.9**	**219.9**	**199.5**	**188.7**	**165.7**	**141.7**	**141.7**	**151.7**	**161.7**
Pension liabilities	2.2	2.9	2.7	3.5	3.6	3.7	3.7	3.8	3.8	3.8	3.9	3.9	3.9	3.9	3.9	3.9	3.9
Provisions	0.7	0.1	0.1	0.0	0.0	0.1	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred tax liabilities	2.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Finance lease liabilities	0.2	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Lease liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	20.4	20.5	20.5	20.5	20.5	20.5	20.5
Other non-current liabilities	2.5	0.9	2.3	2.5	1.7	0.7	0.9	1.3	1.6	1.6	0.7	1.4	1.4	1.4	1.4	1.4	1.4
Deferred income	0.0	0.0	0.0	214.8	97.3	102.5	67.4	23.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Total liabilities	**120.2**	**64.3**	**77.5**	**324.6**	**274.3**	**330.5**	**318.7**	**297.3**	**225.2**	**225.2**	**224.4**	**214.5**	**191.5**	**167.5**	**167.5**	**177.5**	**187.5**
Share capital	154.5	157.3	185.4	223.9	233.4	235.0	235.6	235.7	236.5	236.5	237.3	238.5	238.5	238.5	238.5	238.5	238.5
Share premium account	112.5	114.2	357.4	649.1	993.0	995.3	996.1	1,276.3	1,277.8	1,277.8	1,280.5	1,283.7	2,251.7	2,259.7	2,259.7	2,295.4	2,334.2
Other reserves	0.0	(0.2)	(0.0)	(1.0)	(1.3)	(0.6)	(0.6)	(0.6)	(0.7)	(0.7)	(0.7)	(0.7)	(0.7)	(0.7)	(0.7)	(0.7)	(0.7)
Translation differences	0.2	(1.2)	(0.5)	(1.1)	(1.8)	(1.8)	(1.6)	(1.6)	(1.6)	(1.6)	(1.3)	(1.5)	(1.5)	(1.5)	(1.5)	(1.5)	(1.5)
Accumulated losses	(100.1)	(63.9)	(177.3)	(112.3)	(211.4)	(328.6)	(343.8)	(321.5)	(297.8)	(297.8)	(340.0)	(376.5)	3,122.3	3,053.1	3,053.1	2,768.9	2,515.7
Total stockholders' equity	**167.1**	**206.1**	**365.0**	**758.7**	**1,012.0**	**899.3**	**885.7**	**1,188.2**	**1,214.2**	**1,214.2**	**1,175.8**	**1,143.4**	**5,610.2**	**5,549.0**	**5,549.0**	**5,300.5**	**5,086.1**
Total liabilities and stockholders' equity	**287.4**	**270.5**	**442.5**	**1,083.3**	**1,286.3**	**1,229.9**	**1,204.3**	**1,485.6**	**1,439.5**	**1,439.5**	**1,400.2**	**1,357.8**	**5,801.7**	**5,716.5**	**5,716.5**	**5,478.0**	**5,273.6**

Source: Company data, BCM estimates

Cash flow statement

€ in millions, unless otherwise noted

	2013A	2014A	2015A	2016A	2017A	1Q18A	2Q18A	3Q18A	4Q18A	2018A	1Q19A	2Q19A	3Q19E	4Q19E	2019E	2020E	2021E
Net income (loss)	**(8.1)**	**33.2**	**(118.4)**	**54.0**	**(115.7)**	**(37.3)**	**(21.8)**	**14.8**	**15.0**	**(29.3)**	**(48.7)**	**(47.2)**	**3,498.8**	**(69.2)**	**3,333.8**	**(284.2)**	**(253.2)**
Tax income and expenses	(3.1)	2.3	(1.2)	0.2	0.2	0.1	0.1	(0.5)	0.4	0.1	0.1	(0.1)	0.0	0.0	0.0	0.0	0.0
Other net financial income and expense	0.2	(1.8)	(0.4)	(1.6)	(2.1)	5.2	(12.1)	(2.1)	4.5	(4.4)	(1.6)	1.6	0.0	0.0	0.0	0.0	0.0
Fair value of share subscription agreement	0.0	0.0	30.6	(57.5)	0.0	0.0	0.0	0.0	0.0	0.0	1.5	(1.5)	0.0	0.0	0.0	0.0	0.0
Depreciation and amortization	8.2	4.6	3.4	4.2	4.3	1.2	2.5	1.1	0.3	5.1	2.8	2.9	2.9	2.9	11.4	12.0	13.0
Impairment loss	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.1	1.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net loss on foreign exchange transactions	(2.1)	(0.3)	(0.4)	0.0	0.0	0.1	(0.1)	(0.3)	0.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Share-based compensation	2.7	3.0	5.0	11.0	16.5	3.9	6.6	7.5	8.8	26.8	6.0	10.8	8.0	8.0	32.8	35.8	38.8
Increase or decrease in retirement benefits	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1	0.1	0.1	0.1	0.0	0.0	0.2	0.0	0.0
Gains and losses and other financial expenses	0.0	0.0	0.0	(5.5)	27.5	0.0	0.0	0.0	(10.1)	(10.1)	(4.8)	3.4	0.0	0.0	(1.4)	0.0	0.0
Change in fair value of financial assets	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(1.2)	(1.2)	0.0	2.1	0.0	0.0	2.1	0.0	0.0
Increase or decrease in provisions	(0.1)	0.0	(0.1)	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Increase in pension liabilities	0.2	0.4	0.0	0.0	0.0	0.1	0.1	0.1	(0.2)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gain on disposal of fixed assets	0.0	0.0	(0.1)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gain on sale of service division	0.0	(67.5)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Deferred income	0.0	0.0	0.0	245.8	(65.7)	(34.5)	(25.5)	(33.4)	(59.9)	(153.3)	(26.0)	(27.5)	(23.0)	(24.0)	(100.5)	0.0	0.0
Adjustments for investing and financing	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	(0.7)	(0.7)	0.0	(0.0)	0.0	0.0	(0.0)	0.0	0.0
Interest paid	(0.2)	(0.1)	(0.0)	(0.0)	(0.3)	(0.5)	(0.3)	(0.2)	(0.0)	(1.1)	(0.3)	0.2	0.0	0.0	(0.2)	0.0	0.0
Interest received	1.0	1.0	1.1	1.1	1.3	1.4	1.4	0.5	1.3	4.6	1.6	(1.1)	0.0	0.0	0.4	0.0	0.0
Income taxes paid and received	(0.1)	0.1	(0.1)	(1.8)	(0.2)	0.0	0.0	(0.0)	(0.0)	(0.0)	(0.0)	0.1	0.0	0.0	0.0	0.0	0.0
Changes in working capital																	
Inventory	(0.0)	(0.0)	(0.0)	0.0	0.0	(0.0)	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(10.0)	(10.0)
Receivables	1.1	(10.1)	(7.2)	(13.0)	(27.7)	12.9	(16.1)	(0.1)	3.2	(0.1)	(1.2)	(31.7)	(3.0)	(3.0)	(38.9)	(10.0)	(10.0)
Payables	2.2	(40.3)	(26.7)	2.1	14.8	7.6	13.8	9.0	(10.4)	20.0	(1.1)	18.0	0.0	0.0	17.0	10.0	10.0
Total changes in working capital	**3.3**	**(50.5)**	**(34.0)**	**(10.9)**	**(12.9)**	**20.5**	**(2.3)**	**8.9**	**(7.1)**	**19.9**	**(2.3)**	**(13.6)**	**(3.0)**	**(3.0)**	**(21.9)**	**(10.0)**	**(10.0)**
Cash from operating activities	**1.8**	**(75.6)**	**(114.6)**	**239.4**	**(147.0)**	**(39.8)**	**(51.5)**	**(3.6)**	**(47.5)**	**(142.5)**	**(71.7)**	**(70.0)**	**3,483.7**	**(85.3)**	**3,256.7**	**(246.5)**	**(211.4)**
Purchase of property, plant, and equipment	(7.3)	(2.1)	(6.1)	(4.5)	(5.3)	(1.2)	(1.8)	(1.3)	(6.1)	(10.4)	(2.1)	(2.9)	(108.3)	(1.7)	(115.1)	(11.5)	(14.6)
Purchase of intangible fixed assets	(0.5)	(0.7)	(0.6)	(0.3)	(2.1)	(0.3)	(0.4)	(0.8)	(1.8)	(3.3)	(1.2)	(2.3)	0.0	0.0	(3.5)	0.0	0.0
Proceeds, disposal of intangibles	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0
Proceeds, disposal of PP&E	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Acquisition of financial assets	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(0.2)	0.0	0.0	0.0	(0.2)	0.0	0.0
Proceeds, sale of financial assets	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0
Acquisitions of subsidiaries	(1.2)	0.0	0.0	(2.8)	0.0	0.0	0.0	0.0	(4.6)	(4.6)	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Disposals of subsidiaries	0.0	130.8	0.0	0.0	0.0	0.0	0.0	0.0	2.4	2.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds, available for sale securities					0.4			0.1	(0.1)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Change in restricted cash	(3.0)	(7.4)	2.3	0.2	6.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cash from investing activities	**(12.0)**	**120.6**	**(4.3)**	**(7.3)**	**(0.5)**	**(1.5)**	**(2.2)**	**(1.9)**	**(10.3)**	**(15.9)**	**(3.4)**	**(5.3)**	**(108.3)**	**(1.7)**	**(118.7)**	**(11.5)**	**(14.6)**
Repayments, finance leases and other debts	(0.3)	(0.2)	(0.0)	(0.0)	(0.1)	(0.0)	0.0	0.0	0.0	(0.0)	(1.2)	(0.9)	0.0	0.0	(2.1)	0.0	0.0
Issue cost paid for capital and share premium	0.0	0.0	0.0	(0.3)	(15.8)	0.0	0.0	(15.0)	(1.0)	(16.0)	0.0	7.8	0.0	0.0	7.8	0.0	0.0
Proceeds, exercise of warrants	0.0	0.0	0.0	4.3	5.3	0.0	0.0	5.3	2.4	7.7	3.5	(3.5)	0.0	0.0	0.0	0.0	0.0
Proceeds, capital and share premium inc., net	54.8	4.4	271.4	392.1	363.9	3.9	1.3	290.9	(0.0)	296.2	0.0	0.0	960.0	0.0	960.0	0.0	0.0
Cash from financing activities	**54.5**	**4.2**	**271.4**	**396.0**	**353.4**	**3.9**	**1.3**	**281.2**	**1.4**	**287.9**	**2.2**	**3.4**	**960.0**	**0.0**	**965.7**	**0.0**	**0.0**
Effect of currency rate changes on cash	(0.5)	0.3	0.1	4.8	(27.8)	(5.6)	10.9	1.3	3.5	10.1	5.0	(3.1)	0.0	0.0	1.9	0.0	0.0
Net changes in cash	43.8	49.5	152.6	632.9	178.0	(43.0)	(41.4)	276.9	(52.9)	139.6	(67.9)	(74.9)	4,335.4	(87.0)	4,105.6	(258.0)	(226.1)
Beginning cash and equivalents	94.4	138.2	187.7	340.3	973.2	1,151.2	1,108.2	1,066.8	1,343.7	1,151.2	1,290.8	1,222.9	1,148.0	5,483.4	1,290.8	5,396.4	5,138.4
Ending cash and equivalents	**138.2**	**187.7**	**340.3**	**973.2**	**1,151.2**	**1,108.2**	**1,066.8**	**1,343.7**	**1,290.8**	**1,290.8**	**1,222.9**	**1,148.0**	**5,483.4**	**5,396.4**	**5,396.4**	**5,138.4**	**4,912.3**
Free cash flow	**(5.5)**	**(77.6)**	**(120.7)**	**234.9**	**(152.3)**	**(41.0)**	**(53.3)**	**(4.9)**	**(53.7)**	**(152.9)**	**(73.8)**	**(72.9)**	**3,375.4**	**(87.0)**	**3,141.7**	**(258.0)**	**(226.1)**
FCF/share	€ -0.19	€ -2.58	€ -3.38	€ 4.97	€ -3.08	€ -0.80	€ -1.04	€ -0.09	€ -0.99	€ -2.93	€ -1.35	€ -1.33	€ 54.31	€ -1.39	€ 53.70	€ -4.07	€ -3.51

Source: Company data, BCM estimates

RATING AND PRICE TARGET HISTORY

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Production of the recommendation completed: 09.10.2019, 07:56 GMT

Historical price target and rating changes for Galapagos NV in the last 12 months

Date	Price target - EUR	Rating	First dissemination GMT	Initiation of coverage
December 10, 2018	112.00	Under review	2018-12-11 08:03	March 13, 2018
February 05, 2019	112.00	Buy	2019-02-05 12:04	
April 12, 2019	140.00	Buy	2019-04-12 11:59	
September 10, 2019	200.00	Buy	-	

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SALES TRADING

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