R&D

Research & Development

Pioneering for patients

Our broad pipeline and powerful drug discovery engine

We discover and develop small molecule medicines with novel modes of action, several of which show promising patient results and are currently in late-stage development in multiple diseases with high unmet medical need. Our highly flexible discovery platform is applicable across many therapeutic areas and our pipeline comprises programs ranging from discovery to Phase 3 and registration phase in inflammation, fibrosis, osteoarthritis, and other indications.

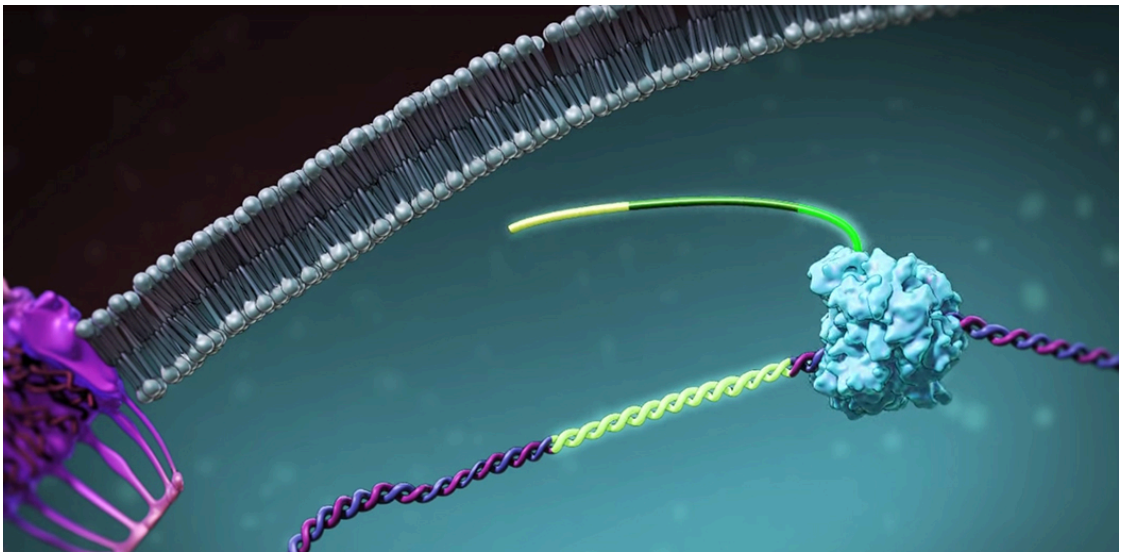
Our clinical pipeline includes: JAK1 inhibitor filgotinib, which is currently filed for approval in RA in the U.S., Europe, and Japan, in Phase 3 trials in UC, CD, and PsA, and in Phase 2 trials in multiple additional indications; autotaxin inhibitor GLPG1690, which is currently in the ISABELA 1 & 2 pivotal trials for idiopathic pulmonary fibrosis (IPF) and the NOVESA Phase 2 proof-of-concept trial in systemic sclerosis (SSc) for which recruitment was completed end of 2019; GLPG1205, a GPR84 inhibitor which completed recruitment in the PINTA Phase 2 proof-of-concept trial in IPF in early 2020; GLPG1972, an ADAMTS-5 inhibitor for which we completed patient recruitment in the ROCCELLA global Phase 2b trial in OA patients in June 2019; and the Toledo molecules GLPG3312, GLPG3970, and GLPG4399, aimed at a novel class of targets we discovered and currently in preclinical and Phase 1 development. Almost exclusively these programs are based on inhibiting targets which were identified using our proprietary target discovery platform.

We have collaborations with Gilead for filgotinib, GLPG1690, and other pipeline assets, with Servier for GLPG1972, with Evotec and Fibrocor for early stage fibrosis programs and with AbbVie in the field of cystic fibrosis (CF). The following table highlights key aspects of our development program indication areas at the beginning of 2020:

Our clinical pipeline

area	preclinical	phase 1	phase 2	phase 3	approval
filgotinib	multiple indications, submitted for RA				
IPF/fibrosis	in ph3 and ph2				
osteoarthritis	ph2b underway				
Toledo	ph1 programs				
inflammation/fibrosis/other	>30 programs				

Flexible target discovery platform



Watch the video on our YouTube channel

Our target discovery platform provides a significant and substantial competitive advantage as it:

- closely mimics the *in vivo* situation through the use of primary human cells with relevant trigger and readout for a specific disease phenotype
- identifies possible points to intervene in a disease pathway by knocking down an individual protein in these pathways; and
- enables us to rapidly analyze all of the druggable genes and select pharmaceutically tractable protein targets directly by their ability to regulate key disease biology

A proof of success of this unique approach is demonstrated with filgotinib which acts on JAK1, a target whose role in the specific disease was discovered by us using our discovery platform. Further proof of this approach was shown in 2017 with autotaxin inhibitor GLPG1690 in IPF patients.

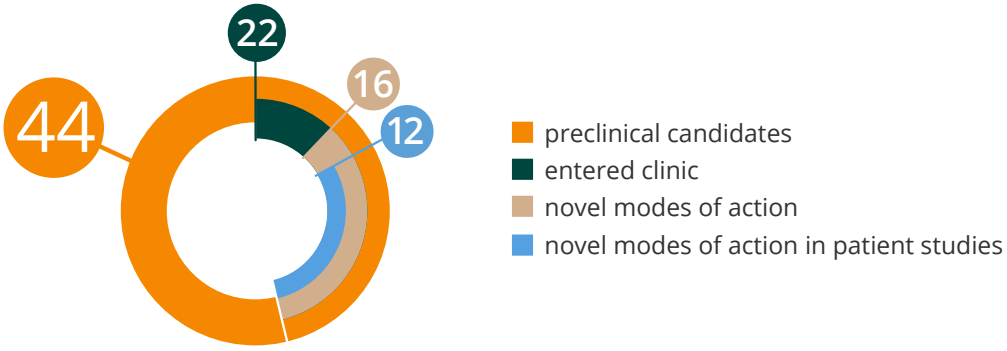
The human genome consists of tens of thousands of genes which code for the proteins that make up the human body. Nearly all chronic diseases and disorders are caused by a disruption in the normal function of certain proteins. The main goal of the industry is to discover and develop molecules that alter the activity of these proteins so that normal function returns and the cause of the disease is minimized or eliminated. One of the main obstacles in discovering new drugs is to understand exactly which of the body's tens of thousands of proteins play a key role in a particular disease. Once these proteins are discovered, they become targets for drug design. Finding these targets is one of the critical steps in the drug discovery process. Our approach to target discovery is unique as our discovery platform focuses on target identification using primary human cells, which we believe provides a good system to study the effect that a protein might have on the disease in the human body.

In order to study proteins in human cells, we take advantage of the distinctive properties of adenoviruses. Adenovirus is the virus that causes the common cold and has the capability to infect almost every type of human cell. The adenoviruses we work with have been engineered to act as a shuttle vehicle, allowing the delivery of specific pieces of DNA into human cells. Additionally, these viruses have been made replication incompetent, meaning they do not replicate in the human cell they infect, and so do not interfere with the processes in the

cell. We engineered the viruses to carry small pieces of DNA, specific for individual human genes. When the virus enters the cell, this DNA piece leads to the production of a short sequence of RNA that is processed in the cell to become "short interfering RNA," or siRNA, which specifically interferes with the mRNA of the protein it was designed for. By using these viruses, we can cause the cells to block, or "knock-down," the production of a certain protein, mimicking what a small molecule drug does in the human body. We built a collection with these adenoviruses, now in excess of 20,000 viruses, that addresses around 6,000 druggable genes.

Our drug discovery research is based on the targets discovered using this technology. Once a target is validated, it is tested against large collections of chemical small molecules to identify chemical structures that interact with the target and block or activate protein production. These chemical structures are then optimized to obtain "drug-like" characteristics followed by testing of the product candidate in the clinic.

This discovery approach provides starting points for the discovery and development of drugs with new modes of action. Since 2009, we have generated 44 preclinical candidates. Of these, 22 have entered first-in-human clinical development, 16 of which have novel modes of action, and 12 entered into patient studies.

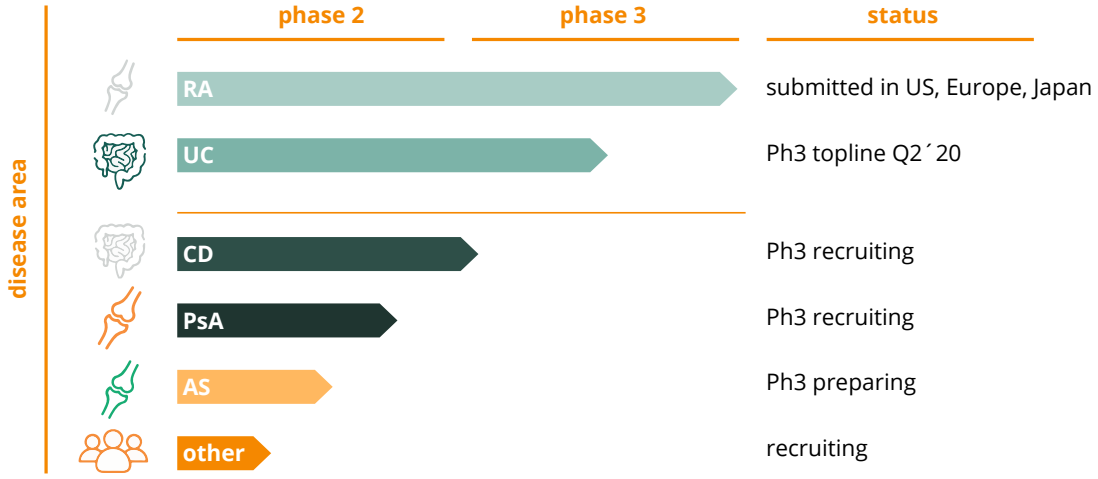


In addition to our pipeline of molecules in the clinic, we have multiple discovery programs which are advancing toward clinical development. Further to targets and molecules in RA, IBD, and fibrosis, we are exploring new modes of action in AS, PsA, IBD, AtD, lupus, IPF, SSc, nonalcoholic steatohepatitis, type 2 diabetes, hepatitis B, osteoarthritis and polycystic kidney disease.

Filgotinib in inflammation

We have a collaboration agreement with Gilead to develop and commercialize filgotinib in multiple diseases. Filgotinib is currently under regulatory review in the United States, Europe, and Japan, and in Phase 3 clinical trials in UC, CD, and PsA, with a Phase 3 in AS expected to start in 2020. Gilead completed trials with filgotinib in Sjögren's disease and cutaneous lupus erythematosus and is working with us to evaluate next steps in those disease areas. In addition, Gilead is running Phase 2 trials with filgotinib in uveitis, small bowel Crohn's disease, and fistulizing Crohn's disease. The following graphic represents the broad filgotinib program. At the time of publication of this report, it was decided to pause the recruitment of ongoing filgotinib trials in connection with the coronavirus pandemic.

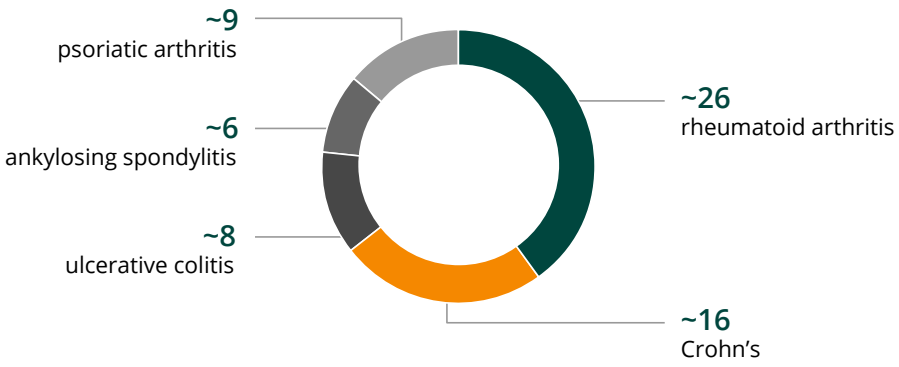
Our filgotinib program



RA: rheumatoid arthritis CD: Crohn's disease UC: ulcerative colitis AS: ankylosing spondylitis PsA: psoriatic arthritis

The market for drugs that treat inflammatory diseases is considerable and growing. We estimate that the inflammation market could grow to approximately $65 billion by 2027, driven by new drugs filling the current unmet need for oral, monotherapy treatments with a rapid response, and higher efficacy maintained over time. RA remains the largest single market indication, which we estimate to be approximately $26 billion, with the other main markets representing a larger combined opportunity than RA:

Inflammation market in ~2027, $B



Source: Galapagos estimates, Decision Resources Group

The Phase 2 and 3 data observed with filgotinib in RA and the Phase 2 data in CD, AS, and PsA thus far, indicate the potential of filgotinib to substantially improve treatment standards in these and other inflammatory conditions. American College of Rheumatology (ACR) scores in Phase 2 and 3 trials in RA patients were significantly greater for filgotinib compared with placebo, and CDAI remission and SES-50 scores are similarly promising with filgotinib in a Phase 2 trial in CD patients who are naive to TNF therapy, and tolerability and safety data were consistently favorable across those trials. Following an interim futility analysis of the Phase 2b/3 SELECTION trial in UC patients, the independent Data Monitoring Committee recommended the trial to proceed into the Phase 3 portion of the study. ACR and enthesitis scores were encouraging with filgotinib in PsA in the EQUATOR Phase 2 trial, while spine mobility and function were significantly improved with filgotinib in AS patients in the TORTUGA Phase 2 trial. Filgotinib is highly selective for JAK1, resulting in favorable tolerability so far, including low rates of infection and low rates of venous thrombotic events (VTEs) reported in all trials.

Filgotinib in RA

RA is a chronic autoimmune disease that affects approximately more than three million patients in the United States and Europe. RA is characterized by inflammation and degeneration of the joints. Patients suffer from pain, stiffness, and restricted mobility due to a persistent inflammation of multiple joints, ultimately resulting in irreversible damage of the joint cartilage and bone. The market for RA treatments in the U.S., EU5 and Japan was worth $28 billion in 2018, with 60% of patients treated with disease-modifying anti-rheumatic drugs (DMARDs), including injectable, biological therapies.[3]

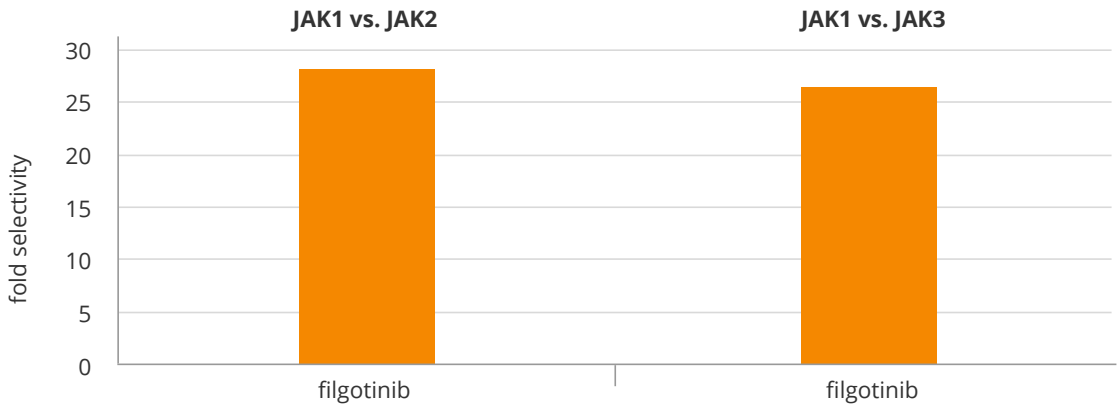
Despite there being many approved agents, considerable unmet need exists, as only one in five patients achieve full remission in the first year.

Oral therapies targeting the Janus kinase (JAK) signaling pathway are approved to treat inflammatory diseases; some JAK inhibitors, however, are associated with a range of side effects, including pulmonary embolisms and aberrations in low-density lipoprotein (LDL, cholesterol) and red blood and NK cell counts. We discovered JAK1 in an inflammation target discovery assay in 2003 and subsequently developed filgotinib as a JAK1 specific small molecule inhibitor. We demonstrated that filgotinib has a nearly 30-fold selectivity for JAK1 over JAK2 and for JAK1 over JAK3. These findings were independently corroborated by Dr. Iain McInnes at the 2017 Annual Meeting of the ACR.

[3] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

Filgotinib selectivity

High selectivity for JAK1



Source: Galapagos human whole blood assay

Source: Galapagos biochemical assay

DARWIN Phase 2b program

We reported positive results from the DARWIN 1 & 2 Phase 2b dose-range finding clinical trials in 2015 and these findings were published in the *Annals of Rheumatological Diseases* (Westhovens *et al.* 2016 and Kavanaugh *et al.* 2016).

DARWIN 3 was a multi-center, open-label, long-term follow-up safety and efficacy trial of subjects who completed either DARWIN 1 or DARWIN 2. All subjects started the trial at the same dose level, either at 200 mg filgotinib once-daily or at 100 mg filgotinib twice per day (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.

We and our collaboration partner Gilead reported findings from DARWIN 3 at 156 weeks of treatment at ACR 2019. The data showed that filgotinib maintained its promising activity levels and that it had a favorable tolerability profile. Data in DARWIN 3 were consistent with the risk/benefit profiles reported in DARWIN 1 and 2, and were presented by Kavanaugh *et al.* at the 2019 Annual Meeting of the ACR.

Below is an overview of selected adverse events for filgotinib observed in DARWIN 3:

event per 100 PYE	filgotinib 50-200 mg DARWIN 3 week 156
patient year exp.	2,203
serious infection	1.0
Herpes zoster	1.5
DVT/ PE	2/2,203* 0.1
deaths	0.2

Data on file; DVT/PE = deep venous thrombosis/pulmonary embolism
* one single patient experiencing DVT and PE

FINCH Phase 3 program

The safety and efficacy of 100 mg and 200 mg filgotinib once daily have been investigated in the FINCH clinical Phase 3 program which was initiated in August 2016 and which includes four Phase 3, randomized, multicenter studies in patients with moderate to severe RA.

The studies were designed to characterize the efficacy and safety of filgotinib in several key patient populations following the typical RA treatment pathway. These included:

- Patients who had an inadequate response to methotrexate (MTX) (FINCH 1)
- Patients with difficult-to-treat RA and an inadequate response to biologic disease-modifying antirheumatic drugs (csDMARD) (FINCH 2)
- Methotrexate-naïve patients (FINCH 3)
- Eligible patients could also roll-over into a long-term extension study (FINCH 4)

In both rat and dog toxicology studies in the preclinical phase, filgotinib induced adverse effects on the male reproductive system. Consequently, Gilead and Galapagos are performing dedicated male patient semen analysis trials in inflammation (RA, CD, UC, AS, and PsA) patients, called MANTA and MANTA-RAy, concurrent to all Phase 3 programs. These randomized, double-blind, placebo-controlled trials are intended to be combined to meet the requirement of 200 adult male inflammation patients with a treatment phase of up to 26 weeks. Recruitment into these trials is, at time of publication of this report, paused in light of the COVID-19 pandemic.

FINCH 1 results

The study achieved its primary endpoint for both doses of filgotinib in the proportion of patients achieving an American College of Rheumatology 20 percent response (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. 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.

Topline FINCH 1 efficacy[4] data are summarized in the table below:

	filgotinib 200 mg +MTX (n=475)[&]	filgotinib 100 mg +MTX (n=480)[&]	adalimumab 40 mg +MTX (n=325)[&]	placebo +MTX (n=475)[&]
ACR20 (%)	76.6***	69.8***	70.8	49.9
ACR50 (%)	47.2***	36.3***	35.1	19.8
ACR70 (%)	26.3***	18.5***	14.2	6.7
DAS28(CRP) ≤ 3.2 (low disease activity) (%)	49.7***[$]	38.8***	43.4	23.4
DAS28(CRP) < 2.6 (clinical remission) (%)	33.9***[¥#]	23.8***[£#]	23.7	9.3
HAQ-DI change	-0.69***	-0.56***	-0.61	-0.42
mTSS change	0.13***	0.17***	0.16	0.38

& Number of patients randomized to each treatment group and who received at least one dose of study drug
ACR20/50/70 represents American College of Rheumatology 20%/50%/70% improvements.
*** p <0.001, compared with placebo
$ p <0.001, non-inferiority to adalimumab
£ p <0.01, non-inferiority to adalimumab
¥ p <0.01, superiority to adalimumab
Comparison not adjusted for multiplicity

FINCH 2 results

Filgotinib achieved its primary endpoint in the FINCH 2 trial in the proportion of patients achieving an American College of Rheumatology 20 percent response (ACR20) at week 12. Also at weeks 12 and 24, the proportion of patients achieving ACR50 and ACR70 response, low disease activity, and clinical remission were significantly higher for patients receiving once-daily filgotinib 100 mg or 200 mg compared to patients receiving placebo. The clinical efficacy and quality of life outcomes assessed at week 12 and week 24 were presented at the Annual ACR meeting 2019 (Genovese *et al.*) and the FINCH 2 results were published in *The Journal of the American Medical Association, JAMA* (Genovese *et al.* 2019).

Topline efficacy data are summarized in the table below:

non-responder imputation	week 12			week 24		
	placebo (n=148)	filgotinib 100 mg (n=153)	filgotinib 200 mg (n=147)	placebo (n=148)	filgotinib 100 mg (n=153)	filgotinib 200 mg (n=147)
ACR20 (%)	31.1	57.5***	66.0***	34.5	54.9***	69.4***
ACR50 (%)	14.9	32.0***	42.9***	18.9	35.3**	45.6***
ACR70 (%)	6.8	14.4*	21.8***	8.1	20.3**	32.0***
DAS28(CRP) < 2.6 (clinical remission) (%)	8.1	25.5***	22.4***	12.2	26.1**	30.6***
DAS28(CRP) ≤ 3.2 (low disease activity) (%)	15.5	37.3***	40.8***	20.9	37.9**	48.3***

ACR20/50/70 represents American College of Rheumatology 20%/50%/70% improvements.
* p <0.05, compared to placebo
** p <0.01, compared to placebo
*** p <0.001, compared to placebo

[4] All efficacy time points assessed at Week 12 except mTSS which was assessed at Week 24

FINCH 3 results

The study achieved its primary endpoint in the proportion of patients achieving an American College of Rheumatology 20 percent response (ACR20) at week 24. The proportion of patients achieving the primary endpoint of ACR20 response at week 24 was significantly higher for filgotinib 200 mg plus MTX and filgotinib 100 mg plus MTX compared with MTX alone.

The proportion of patients achieving ACR50, ACR70, and clinical remission (DAS28(CRP) < 2.6) at week 24 was also significantly higher for patients receiving once-daily filgotinib 100 mg or 200 mg plus MTX compared with patients receiving MTX alone. Additionally, those who received filgotinib experienced greater reduction in the Health Assessment Questionnaire Disability Index (HAQ-DI) compared with those receiving MTX alone at week 24. Filgotinib 200 mg monotherapy inhibited the progression of structural damage at week 24 compared with MTX alone as assessed by modified total Sharp score (mTSS).

Topline FINCH 3 efficacy[5] data are summarized in the table below:

	filgotinib 200 mg +MTX (n=416)[&]	filgotinib 100 mg +MTX (n=207)[&]	filgotinib 200 mg monotherapy (n=210)[&]	MTX (n=416)[&]
ACR20 (%)	81.0***	80.2*	78.1	71.4
ACR50 (%)	61.5***	57.0**	58.1**[#]	45.7
ACR70 (%)	43.8***	40.1***	40.0***[#]	26.0
DAS28(CRP) < 2.6 (clinical remission) (%)	54.1***	42.5***	42.4***[#]	29.1
HAQ-DI change	-0.94***	-0.90**	-0.89*[#]	-0.79
mTSS change	0.20	0.22	-0.04**[#]	0.52

MTX, methotrexate
& Number of patients randomized to each treatment group and who received at least one dose of study drug
ACR20/50/70 represents American College of Rheumatology 20%/50%/70% improvements.
* p < 0.05 compared with MTX
** p <0.01, compared with MTX
*** p <0.001, compared with MTX
# Comparison not adjusted for multiplicity

FINCH safety data

We and Gilead also announced interim safety information from four studies of the investigational compound filgotinib for the treatment of rheumatoid arthritis. The data include 24 week results of the Phase 3 FINCH 1, 2, and 3 trials in patients with RA and the pooled analyses from these 3 FINCH trials were presented at the Annual ACR meeting 2019 (Winthrop *et al*). In this pooled analysis, filgotinib was well-tolerated, no new safety concerns were identified, and the safety results were consistent with selective JAK1 inhibition. Adverse events of MACE and DVT/PE were rare and occurred in similar number among all treatment groups. Herpes zoster reactivation was not increased in the filgotinib groups compared with the other treatment groups. The data highlight the favorable safety and tolerability profile of filgotinib as monotherapy and in conjunction with MTX/csDMARD in RA.

[5] Efficacy assessed at Week 24 for all endpoints

Week 24 safety data from the FINCH 1, 2, and 3 studies are aggregated and summarized in the table below. Data from 3,452 patients are reported, including 2,088 patients who received filgotinib:

	Placebo/ csDMARD	adalimumab	filgotinib	filgotinib	filgotinib	filgotinib
			100 mg	200 mg	200 mg	total
		+MTX 40 mg EOW	+MTX/ csDMARD	+MTX/ csDMARD		
	(n=1039) no. (%)	(n=325) no. (%)	(n=840) no. (%)	(n=1038) no. (%)	(n=210) no. (%)	(n=2088) no. (%)
serious infections[&]	10 (1.0)	8 (2.5)	13 (1.5)	13 (1.3)	3 (1.4)	29 (1.4)
Herpes zoster[&]	4 (0.4)	2 (0.6)	5 (0.6)	6 (0.6)	1 (0.5)	12 (0.6)
DVT/PE[&]	3 (0.3)	0 (0)	0 (0)	1 (0.1)[μ]	0 (0)	1 (<0.1)
death[@]	2 (0.2)	0 (0)	1 (0.1)	3 (0.3)	0 (0)	4 (0.2)
malignancy excluding NMSC[&]	4 (0.4)	1 (0.3)	1 (0.1)	0 (0)	0 (0)	1 (<0.1)
MACE[&]	5 (0.5)	1 (0.3)	2 (0.2)	2 (0.2)	1 (0.5)	5 (0.2)

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

& Treatment-emergent events

μ Excludes one retinal vein occlusion

@ All events

Applications for approval of filgotinib in RA

Gilead announced acceptance of a Marketing Authorisation Application (MAA) by the European Medicines Agency in August 2019, submission of a New Drug Application (NDA) to the Japanese Ministry of Health, Labor, and Welfare (MHLW) in October 2019, and submission of an NDA (under priority review) to the United States Food & Drug Administration (FDA) in December 2019. We and our collaboration partner Gilead expect decisions on potential approvals in all these geographies in the course of 2020.

Commercialization of filgotinib in RA

If approved by the European Commission for RA indications, we expect to launch commercial sales activities in Belgium, The Netherlands, and Luxembourg where we are solely responsible for commercialization, and in France, Italy, and Spain where we will lead commercial sales responsibilities in RA, pursuant to the parties' joint commercialization of filgotinib in those countries. We are advanced in our preparations to launch in these countries in the course of 2020, pending approval of filgotinib. Gilead will launch commercial sales activities in RA in Germany and the UK, the remaining of the eight countries in which we and Gilead will equally split profits from filgotinib commercial activities, pursuant to the parties' joint commercialization of filgotinib in those countries. Gilead will be responsible for the commercial launches in all territories outside these eight European countries, should filgotinib be approved in these territories. See details on the Gilead collaboration in the Notes to the consolidated financial statements.

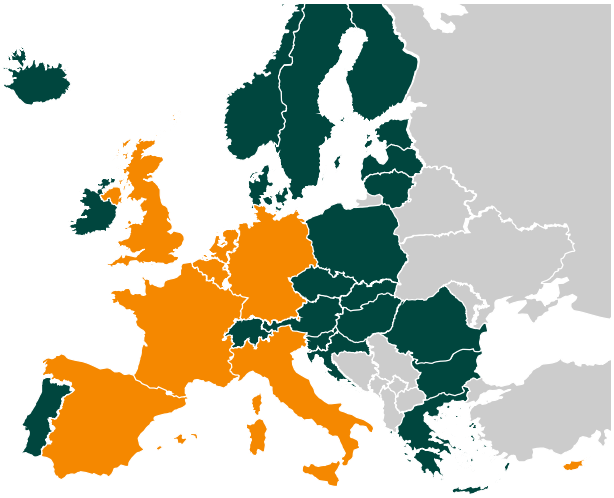
European commercial footprint

2020 – 2021 filgotinib

- Benelux
- France, Italy, Spain
- UK, Germany

2022 – 2023

- Roll out in rest of Europe
- Future products



Filgotinib in IBD, which includes UC and CD

Current treatments for IBD are dominated by anti-TNF agents, with new biologic agents gaining market share.

We observed high activity and a favorable tolerability profile in a Phase 2 trial with filgotinib in CD, as reported in *The Lancet* (Vermeire *et al.* 2016). The profile we saw with filgotinib in this CD patient trial indicates that the product candidate may show activity and tolerability in UC patient trials as well.

Should filgotinib be approved commercially for IBD indications, Galapagos will be lead commercial sales responsible for the UK, Germany and the Benelux countries, and Gilead will be lead commercial sales responsible for France, Italy and Spain. All other countries will be Gilead's commercial sales responsibility.

Global SELECTION Phase 2b/3 program in UC

UC is an inflammatory bowel disease resulting in ulcerations and inflammation of the colon and rectum. In 2018, nearly 2 million patients were diagnosed with UC in the U.S., EU5 and Japan, and the total market for UC treatments in the acute and maintenance settings was worth $6 billion in the U.S., EU5 and Japan in 2018.[6]

Although the introduction of anti-TNF biologics has improved the treatment of some patients, only 33% of patients will achieve long-term remission, and many patients lose their response to treatment over time. The medical need for improved efficacy is high.

Gilead initiated the global SELECTION Phase 2b/3 trial in UC with filgotinib in December 2016. SELECTION investigates efficacy and safety of 100 mg and 200 mg filgotinib once-daily compared to placebo in 1,300 patients with moderately to severely active disease including those with prior antibody therapy failure. Men and women in SELECTION were randomized to receive placebo, 100 mg or 200 mg filgotinib. Due to preclinical findings with filgotinib regarding semen parameters, in the United States, males may receive 200 mg if they failed at least one anti-TNF therapy and vedolizumab, a monoclonal anti-integrin antibody marketed by Takeda. Adjacent to the filgotinib Phase 3 programs, we and Gilead are conducting dedicated male semen analysis studies in CD and UC patients (MANTA) and in RA, PsA, and AS patients (MANTA-RAy).

[6] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

In May 2018, Gilead and we announced that an independent Data Monitoring Committee (DMC) conducted a planned interim futility analysis of SELECTION after 350 patients completed the induction period in the Phase 2b portion of the trial. The DMC recommended that the study could proceed into Phase 3 as planned at both the 100 mg and 200 mg once-daily dose level in biological-experienced and biological-naive patients.

Gilead announced completion of recruitment for SELECTION in 2019, and topline results are expected in the second quarter of 2020.

FITZROY Phase 2 and global DIVERSITY Phase 3 program in CD

CD is an IBD of unknown cause, resulting in chronic inflammation of the gastrointestinal (GI) tract with a relapsing and remitting course. In 2018, nearly 1.5 million patients were diagnosed with CD in the U.S., EU5 and Japan, and the total market for CD treatments in the acute and maintenance settings was worth $16 billion in the U.S., EU5 and Japan in 2018.[7]

Today, only 10% of CD patients on treatment achieve prolonged clinical remission. There are currently no highly effective oral therapies approved for CD and, similar to RA, treatment is dominated by injectable, biological treatments including anti-TNF therapies. Anti-TNF agents have improved the management of CD; however, not all patients respond to these drugs, and secondary loss of response is reported in up to 50% of patients per year in placebo-controlled trials. There continues to be a considerable unmet need with these existing treatments. Dysregulation of the JAK signaling pathway has also been associated with CD, and this suggests that filgotinib, with its high selectivity for JAK1, is a highly attractive candidate for the treatment of CD. It is hypothesized that by inhibiting JAK1, unwanted effects such as anemia may be reduced. This is of particular importance to IBD patients, who frequently experience fecal blood loss.

Our FITZROY Phase 2 trial evaluated the efficacy and safety of once-daily filgotinib in 174 patients with moderate to severe active CD and mucosal ulceration. Patients recruited were either anti-TNF naive or anti-TNF failures. As reported in *The Lancet* (Vermeire *et al.* 2016), the FITZROY trial achieved the primary endpoint of clinical remission at week 10 and filgotinib demonstrated a favorable tolerability profile consistent with the DARWIN trials in RA.

Gilead initiated the Phase 3 DIVERSITY trial with filgotinib in CD in November 2016. The DIVERSITY Phase 3 trial investigates the efficacy and safety of 100 mg and 200 mg filgotinib once-daily compared to placebo in patients with moderate to severe active disease including those with prior antibody therapy failure. Gilead will recruit approximately 1,300 patients from the United States, 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. Due to preclinical findings with filgotinib regarding semen parameters, in the United States, males may receive 200 mg if they failed at least one anti-TNF therapy and vedolizumab. Adjacent to the filgotinib Phase 3 programs, we and Gilead are conducting dedicated male semen analysis studies in CD and UC patients (MANTA) and in RA, PsA, and AS patients (MANTA-RAy). At the time of publication of this report, it was decided to pause recruitment for DIVERSITY in connection with the corona virus pandemic.

In March 2017, Gilead initiated a Phase 2 trial in small bowel CD and a Phase 2 trial in fistulizing CD. Recruitment for these studies has also been paused in connection with the corona virus pandemic.

[7] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

Filgotinib in psoriatic arthritis

PsA is an inflammatory form of arthritis, affecting up to 30% of psoriasis patients. In 2018, 3.5 million patients suffered from PsA in the U.S., EU5 and Japan and the market for PsA treatments was worth nearly $7 billion in 2018 in these seven major markets.[8]

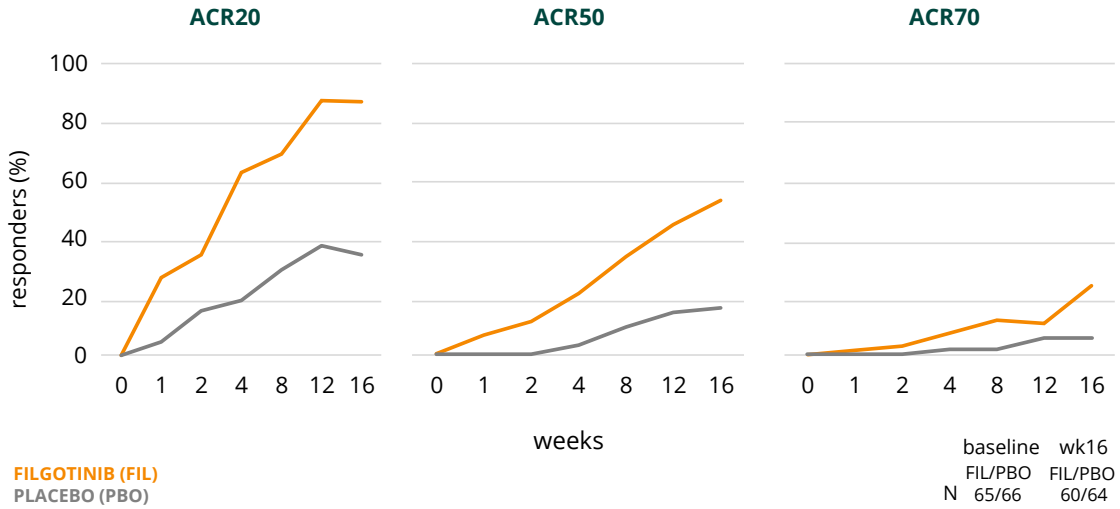
PsA can cause swelling, stiffness and pain in and around the joints and cause nail changes and overall fatigue. Studies show that delaying treatment for PsA as little as six months can result in permanent joint damage. Early recognition, diagnosis and treatment of PsA are critical to relieve pain and inflammation and help prevent joint damage. Despite the availability of a number of treatment options, few current treatments effectively relieve the enthesitis (inflammation of the tendons or ligaments) and symptoms in the joints and the skin.

EQUATOR Phase 2 program with filgotinib in PsA

The EQUATOR Phase 2 trial was a multi-center, randomized, double-blind, placebo-controlled trial to assess the safety and efficacy of filgotinib in adult patients with moderate to severe active PsA. 131 patients were randomized in the trial in a 1:1 ratio to receive 200 mg filgotinib or placebo once-daily administered for 16 weeks. EQUATOR was recruited in eight European countries.

In May 2018, Gilead and we announced that the EQUATOR trial achieved its primary endpoint of improvement in the signs and symptoms of PsA at Week 16, as assessed by ACR20 score. There was an ACR20 response of 80% for filgotinib versus 33% for placebo (p<0.001). The ACR50 and ACR70 responses at week 16 were also significantly higher for filgotinib versus placebo (ACR50: 48% for filgotinib versus 15%, p<0.001; ACR70: 23% versus 6%, p<0.01).

Durable response in EQUATOR PsA Ph2

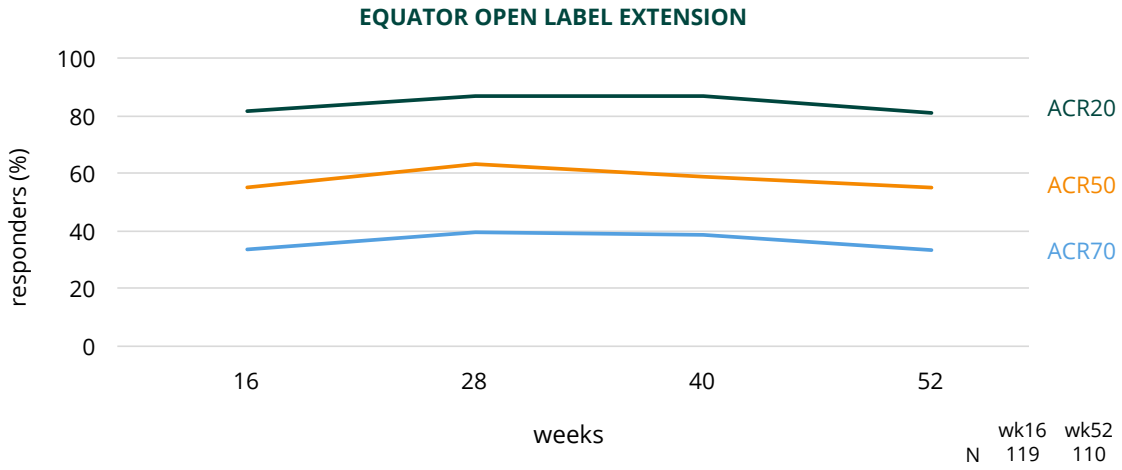


Source: Coates *et al.* ACR 2019

[8] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

This efficacy response was sustained in the open label extension of EQUATOR, up to 52 weeks:

Durable response in EQUATOR PsA Ph2



Source: Coates *et al.* ACR 2019

Filgotinib was generally well-tolerated in the EQUATOR trial, with no new safety signals observed and similar laboratory changes compared to those reported in previous trials with filgotinib in RA patients. The adverse event rate was similar in both groups with mostly mild or moderate events reported. There was one serious infection in the filgotinib group, a patient who experienced pneumonia with a fatal outcome. One other patient receiving filgotinib developed herpes zoster. There were no cases of opportunistic infection, tuberculosis, thromboembolism, or malignancy. The full results of EQUATOR were published in *The Lancet* and presented in a plenary session at ACR 2018 (Mease *et al.* 2018), and a safety update through 52 weeks was presented at ACR2019 (Coates *et al.* 2019).

TEAEs of special interest	incidence # of pts (%) FIL 200 mg, n=65 wk 0-16	incidence # of pts (%) placebo, n=66 wk 0-16	rate/100 PYE # of events FIL 200 mg, PYE=160 wk 0-52
all serious infections	1 (1.5)	–	1.9 (3)
opportunistic infections	–	–	–
herpes zoster	1 (1.5)	–	0.6 (1)
malignancies	–	–	0.6 (1)
deep vein thrombosis	–	–	–
pulmonary embolism	–	–	–
major cardiac events (adjudicated)	–	–	0.6 (1)
deaths	1 (1.5)	–	0.6 (1)

Global PENGUIN Phase 3 program with filgotinib in PsA

In December 2019, Gilead dosed the first patient in the PENGUIN Phase 3 program in PsA. The PENGUIN program investigates the efficacy and safety of 100 mg and 200 mg filgotinib once-daily compared to placebo. PENGUIN 1 will compare the efficacy and safety of filgotinib, adalimumab, and placebo in approximately 1000 patients with active PsA who are naive to bDMARD therapy. PENGUIN 2 will measure efficacy and safety of filgotinib vs placebo

in 390 patients with active PsA who have an inadequate response or are intolerant to bDMARD therapy. The primary endpoint of each trial is ACR20 response at Week 12, with multiple secondary endpoints on signs and symptoms of PsA up to week 24 in PENGUIN 1, and week 16 in PENGUIN 2.

Other indications with filgotinib

Ankylosing spondylitis (AS)

AS, a systemic, chronic, and progressive inflammatory arthritis, is one of the most common rheumatic diseases across the globe, affecting nearly 2 million patients in the U.S., Europe, and Japan in 2018. The total market for AS treatments was worth $3 billion in 2018 in the seven major markets.[9]

AS primarily affects the spine and sacroiliac joints and progresses into severe inflammation that fuses the spine, leading to permanent painful stiffness of the back. Currently, there is no known cure for AS, but there are treatments and medications available to reduce symptoms and manage pain. Recent studies show that the newer biologic medications can potentially slow disease progression in some patients; however, patients respond to different medications with varying levels of effectiveness. Thus, it takes time to find the most effective course of treatment.

TORTUGA was a multi-center, randomized, double-blind, placebo-controlled, Phase 2 trial to assess the safety and efficacy of filgotinib in adult patients with moderate to severe active AS. The trial was conducted in Belgium, Bulgaria, Czech Republic, Estonia, Poland, Spain and Ukraine. In total, 116 patients were randomized in a 1:1 ratio to receive filgotinib 200 mg or placebo once-daily for 12 weeks.

In September 2018, Gilead and we announced that the TORTUGA trial achieved its primary efficacy endpoint in adults with moderately to severely active AS. In the trial, patients treated with filgotinib achieved significantly greater improvements in AS Disease Activity Score, the primary endpoint, at week 12, with a mean change from baseline of -1.5 versus -0.6 for those treated with placebo ($p<0.0001$). More patients receiving filgotinib also achieved an Assessment in AS Response of at least 20% improvement compared to those treated with placebo (76% versus 40%, $p<0.0001$).

Adverse events were generally mild or moderate in severity and were reported in an equal proportion of patients in the filgotinib and placebo groups. Laboratory changes were consistent with those previously reported for filgotinib, and no new safety signals were observed in the trial. There was one treatment-emergent serious adverse event reported for a patient receiving filgotinib who experienced pneumonia and recovered after hospital-based antibiotic treatment. One patient randomized to filgotinib, with an inherited risk for thrombosis, experienced a non-serious deep venous thrombosis after completing the course of study drug. No deaths, malignancies, hepatic events, opportunistic infections or cases of herpes zoster were observed in the study. The full results of the TORTUGA trial were reported in *The Lancet* (Van der Heijde *et al.* 2018).

We expect that our collaboration partner Gilead will initiate a Phase 3 program with filgotinib in AS during the course of 2020.

Additional indications

In the course of 2017, Gilead initiated clinical trials with filgotinib in Sjögren's disease, cutaneous lupus erythematosus, lupus membranous nephropathy, and uveitis. In 2019, Gilead reported completion of the trials in Sjögren's disease and cutaneous lupus erythematosus, and that they are no longer recruiting for lupus membranous nephropathy.

[9] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

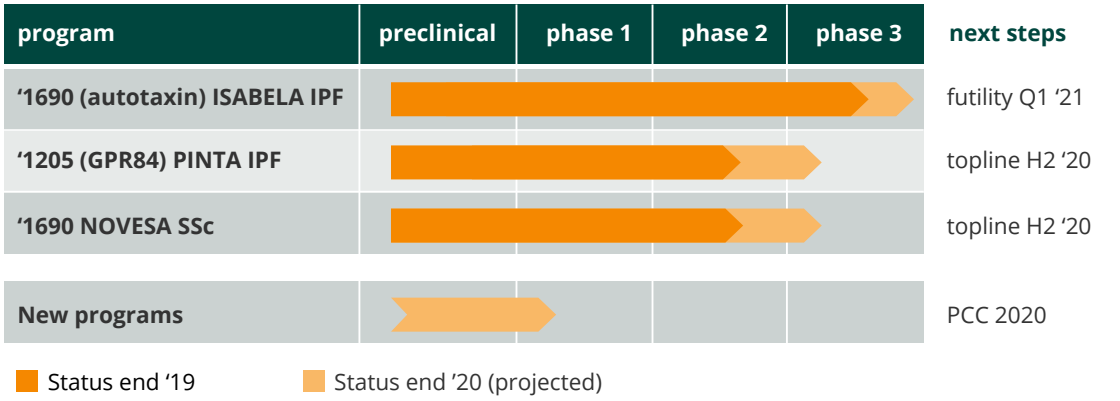
Our fibrosis portfolio

We are building a fibrosis portfolio with different modes of action, with an initial focus on IPF and aim to expand to other forms of organ and skin fibrosis. To this end, we are currently working on a number of drug candidates with distinct novel mechanisms of action, which are fully proprietary to us. In IPF, we believe that having multiple mechanisms of action within our own portfolio of candidates allows the exploration of combinations of therapies. Last year we expanded clinical research into SSc, and we plan to explore additional fibrotic indications with our earlier stage compounds. At the time of publication of this report, it was decided to temporarily pause the start of Phase 1 studies, given de COVID-19 pandemic.

Our IPF portfolio and expected clinical development in 2020:

Building an IPF & fibrosis portfolio

Two toplines in H2 '20, ISABELA futility in Q1 '21



About IPF

IPF is a chronic, relentlessly progressive fibrotic disorder of the lungs that typically affects adults over the age of 40. In 2018, 232,000 patients were diagnosed with IPF in the U.S., EU5 and Japan,[10] and this population is expected to grow, in part thanks to improved diagnosis. Furthermore, prevalence is expected to increase with the aging population and worsening air pollution. The clinical prognosis of patients with IPF is poor, as the median survival at diagnosis is two to four years. Currently, no therapies have been found to cure or stop the progression of IPF. The current treatment strategy aims to slow disease progression and improve quality of life. Lung transplantation may be an option for appropriate patients with progressive disease and minimal comorbidities.

Regulatory agencies have approved Esbriet (marketed by Roche/Genentech) and Ofev (marketed by Boehringer Ingelheim) for the treatment of mild to moderate IPF. Both Esbriet and Ofev have been shown to slow the rate of functional decline in IPF and are gaining ground as the standard of care worldwide. Combined sales of both drugs reached $2.1 billion in 2018.[11] These regulatory approvals represent a major breakthrough for IPF patients; yet neither drug stops the decline in lung function, and the disease in most patients on these therapies continues to progress. Moreover, the adverse effects associated with these therapies are considerable (e.g., diarrhea and liver function test abnormalities with Ofev; nausea and rash with Esbriet). Therefore, there is still a large unmet medical need as IPF remains a major cause of morbidity and mortality.

[10] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

[11] Sales figures from Roche (pirfenidone; Esbriet®) and Boehringer Ingelheim (nintedanib; Ofev®)

We estimate that the market of approved IPF drugs could grow to $5 billion by 2025.

Our IPF trials

GLPG1690

Our most advanced IPF asset is our product candidate GLPG1690, a potent and selective inhibitor of autotaxin (ATX), for which Gilead in-licensed ex-European rights in July 2019 and which is currently in Phase 3.

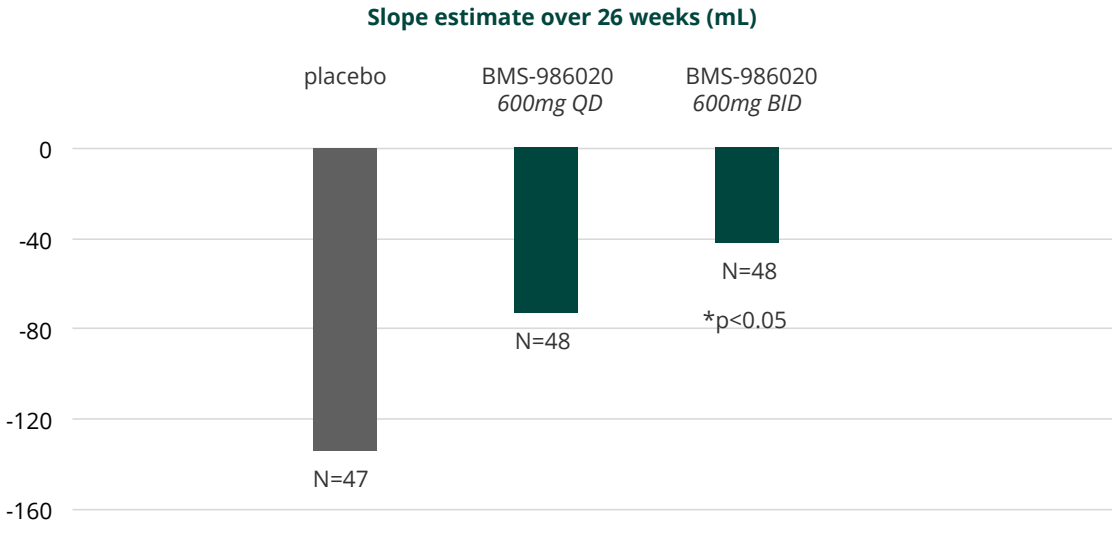
We have received orphan drug designation for GLPG1690 in IPF from the FDA and the European Commission.

We identified ATX as a potential target for IPF, after finding the target using an inflammation assay in our target discovery platform. We evaluated GLPG1690 in a preclinical lung fibrosis model (bleomycin-treated mice) and observed effects on reducing the fibrotic score, numerically favoring GLPG1690 over Esbriet.

Pharmacology and translational studies published by other parties since then suggest that ATX may also play a role in metabolic disease, arthritic pain, oncology, and lung disease. A publication by Palmer *et al.* published in *Chest* in 2018 on the Phase 2 trial data with BMS-986020, a high-affinity LPA1 antagonist developed by Bristol Meyers Squib, showed that BMS-986020 had activity in reducing loss of Forced Vital Capacity in mL (FVC) in IPF patients. LPA1 acts downstream of autotaxin in the biology of IPF, supporting further evaluation of ATX inhibition.

In the course of 2019, BMS published data from the Phase 2 trial with BMS-986020 demonstrating that this compound slowed the rate of FVC decline in a dose-dependent manner, with significance versus placebo. The study was terminated due to off-target effects linked to the compound. However, the reduction in slope estimate over 26 weeks (shown below) indicates that this pathway may be effective in impacting the course of IPF and further validates our approach with GLPG1690.

BMS validation of ATX pathway in patients

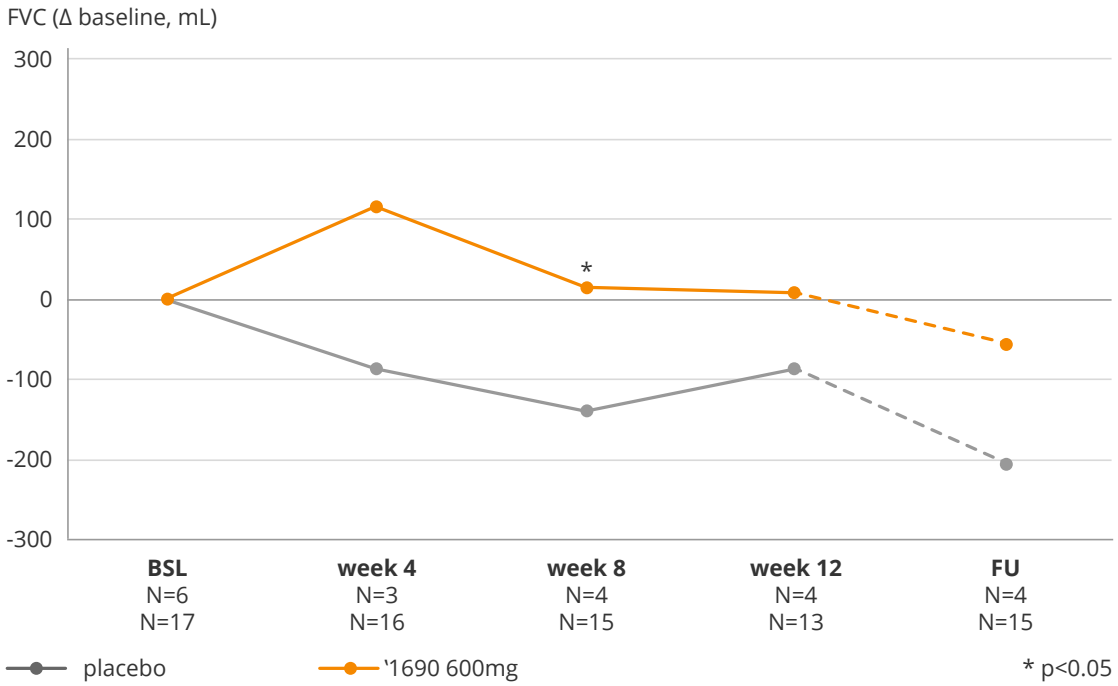


In August 2017, we announced positive topline results for our Phase 2a FLORA trial in IPF patients. This randomized, double-blind, placebo-controlled trial in 23 IPF patients investigated a once-daily 600 mg oral dose of GLPG1690 or placebo of whom 17 received GLPG1690 and six received placebo. The primary objectives of the trial

included the assessment of safety, tolerability, pharmacokinetics and pharmacodynamics of GLPG1690 in an IPF patient population. Secondary objectives included the evaluation of lung function, changes in disease biomarkers, functional respiratory imaging (FRI), and quality of life. The IPF diagnosis was confirmed by central reading.

Over the 12-week period, patients receiving GLPG1690 showed an FVC increase of 8 mL, while patients on placebo showed an FVC reduction of 87 mL (mean from baseline):

FVC: stabilization by '1690

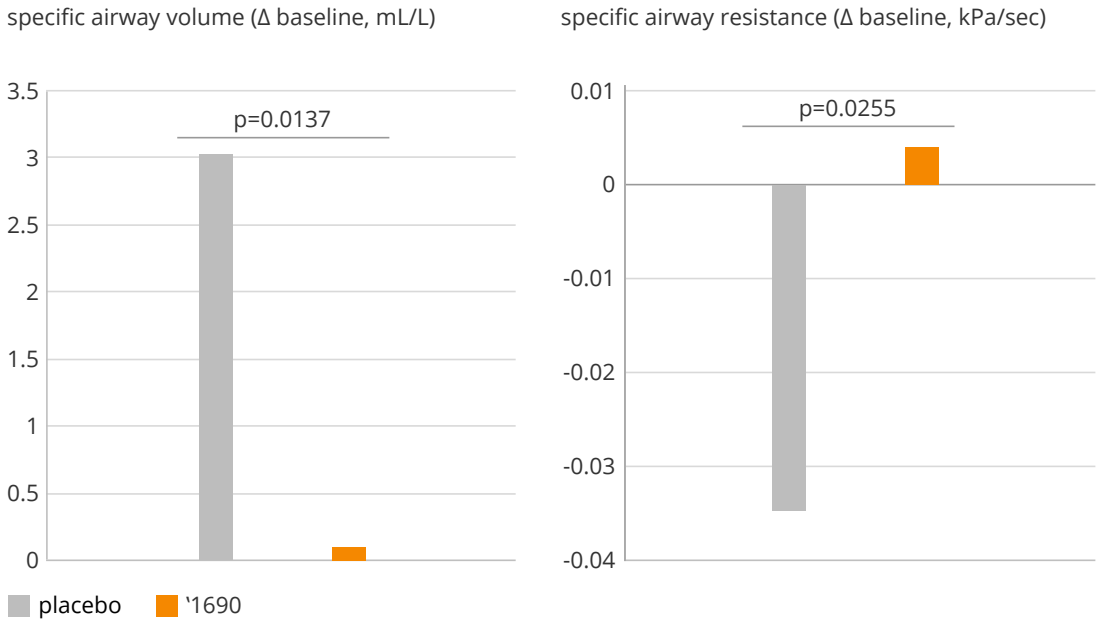


FU: follow-up

In addition to the demonstrated absence of lung function decline over the 12-week period, more sensitive FRI confirmed disease stabilization in the GLPG1690 arm, versus disease progression in the placebo arm, reaching nominal statistical significance on two specific parameters, despite the trial not being powered for significance:

FRI: airway volume & resistance

Significant difference between '1690 & placebo



Patients on GLPG1690 treatment showed a clear reduction of serum LPA18:2, a biomarker for autotaxin inhibition, as expected based on the mechanism of action of GLPG1690. Thus, the level of target engagement observed in Phase 1 with healthy volunteers was confirmed in IPF patients in FLORA.

GLPG1690 was found to be generally well-tolerated in this Phase 2 FLORA trial. Rates of discontinuation due to adverse events, as well as serious adverse event rates, were similar between patients on GLPG1690 and placebo.

The full FLORA results were published in *The Lancet Respiratory* (Maher *et al.* 2018).

Following the encouraging results from the FLORA trial, in 2018 we announced the design of our worldwide Phase 3 program, ISABELA, based on feedback from the FDA and EMA. The ISABELA Phase 3 program consists of two identically designed trials, ISABELA 1 & 2, and plan to enroll a total of 1,500 IPF patients combined. Recruitment will be worldwide, with a significant proportion of patients in the U.S. and Europe. The program is intended to support application for a broad label in IPF in both the NDA and Market Authorization Application (MAA) submissions in, respectively, the U.S. and EU. Patients continue on their standard of care and are randomized to one of two doses of GLPG1690 or placebo. The primary endpoint is the rate of decline of FVC (in mL) until week 52. Secondary assessments include respiratory-related hospitalizations, mortality, quality of life, safety and tolerability.

All patients will continue on their treatment until the last patient in their respective trial has completed 52 weeks of treatment. Therefore, some patients will remain in the study for substantially longer than 52 weeks. This approach will allow assessment of less frequent clinical events that are otherwise difficult to assess in conventional clinical studies of one-year duration.

The following is an overview of the ISABELA trial design:

Phase 3 program ISABELA 1&2

- 1,500 IPF patients total in two identical Phase 3 studies
- Patients remain on standard of care throughout
- Global program with U.S. & EU component
- Primary endpoint: FVC at Week 52
- Secondary endpoints: hospitalizations, mortality, quality of life, safety/tolerability

First patient dosing in ISABELA was announced in December 2018, and as of early 2020, nearly all centers were opened and >800 patients were randomized. We announced that a futility analysis for the ISABELA program is expected to read out in Q1 2021.

Since closing of our collaboration agreement with Gilead in 2019, Galapagos and Gilead share the costs for ISABELA 1 & 2. Galapagos will be responsible for commercial sales of GLPG1690 in Europe, should the candidate be approved; Gilead will be responsible for all commercial activities ex-Europe. See also further details on the Gilead collaboration in the Notes to the consolidated financial statements.

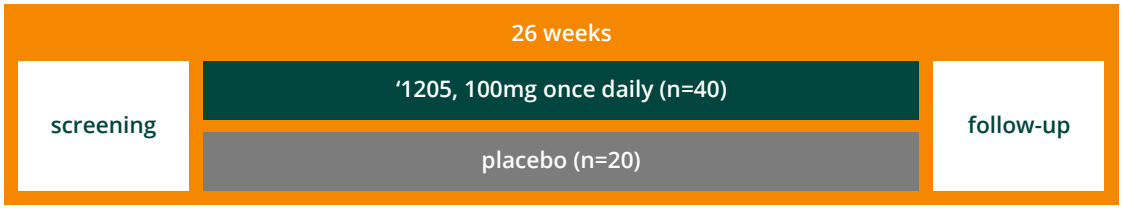
GLPG1205

The second product candidate for IPF in our pipeline is GLPG1205, currently in a Phase 2 trial called PINTA.

GLPG1205 is a small molecule selectively inhibiting GPR84, a target discovered by us. GLPG1205 showed a reduction in signs and symptoms in IPF animal models and has shown favorable tolerability in healthy volunteers and UC patients in previous trials.

PINTA is a randomized, double-blind, placebo-controlled trial investigating a 100mg once-daily oral dose of GLPG1205. The drug candidate or placebo will be administered for 26 weeks in up to 60 IPF patients. Patients may remain on their local standard of care as background therapy. The primary objective of the trial is to assess the change from baseline (FVC in mL over 26 weeks compared to placebo. Secondary measures include FRI, safety, tolerability, pharmacokinetics and pharmacodynamics, time to major events, changes in functional exercise capacity, and quality of life. IPF diagnosis will be confirmed by central reading. Recruitment for PINTA took place in Europe and the Middle East.

PINTA Phase 2 in IPF



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

The first patient dosing was announced in October 2018, and recruitment was completed in early 2020, with topline results from this trial expected in H2 2020.

Our fibrosis trials

Systemic sclerosis (SSc)

SSc is a severe autoimmune disease. One of the most visible manifestations is hardening of the skin. In 2018, 135,000 patients were diagnosed with SSc in the U.S., EU5 and Japan.[12]

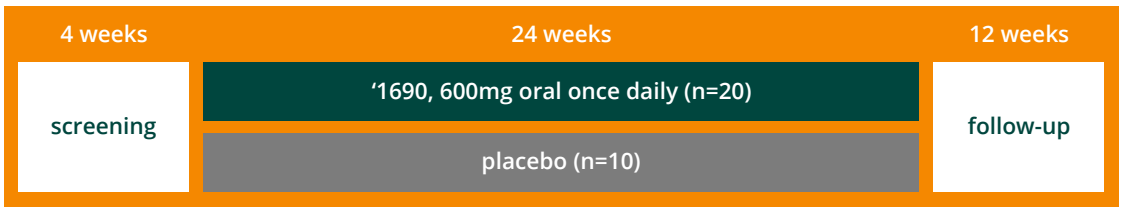
Broadly speaking, there are two types of SSc: limited cutaneous SSc, where skin involvement is limited, and diffuse cutaneous SSc. In diffuse cutaneous SSc, which represents about 35% of the SSc patient population, skin thickening affects several body parts, and patients have a higher risk of developing fibrosis of various internal organs, such as the lung. SSc has one of the highest mortality rates among rheumatic diseases.

Currently, there are no approved disease-modifying drugs to treat this disease. Hence, SSc represents a significant unmet medical need. Current standard of care mainly consists of immunosuppressive drugs and other symptom-alleviating therapies such as methotrexate or cyclophosphamide, and aims to avoid cutaneous fibrosis, interstitial lung disease and renal crisis.

Early 2019, we initiated the NOVESA trial, a double-blind, placebo-controlled Phase 2a trial evaluating the efficacy, safety and PK/PD of GLPG1690 in up to 30 patients with diffuse cutaneous SSc.

We have received orphan drug designation for GLPG1690 in SSc from the FDA and the European Commission.

NOVESA Phase 2a in SSc



[12] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

- 30 patients with progressive diffuse (multi-organ) SSc
- Recruitment in U.S. & 5 EU countries
- Primary endpoint: modified Rodnan Skin Score at week 24
- Secondary & exploratory endpoints: safety, tolerability, broad range of measures (FVC, QoL, CRISS)

The primary endpoint of NOVESA is the modified Rodnan skin score (mRSS) at week 24. The mRRS measures the skin thickness as a surrogate measure of disease severity and mortality, with an increase in thickness associated with involvement of internal organs and increased mortality. Secondary objectives and exploratory endpoints include FVC, quality of life, and other scores.

We completed recruitment for NOVESA in December 2019 and expect topline results in H2 2020.

Our fibrosis partnerships further strengthen the fibrosis pipeline

In January 2019, we announced a global collaboration with Fibrocor focused on a small molecule inhibitor program (in the lead optimization phase) against a novel target for IPF and other indications. We are responsible for the further development and commercialization of the program. In January 2020, we further expanded our collaboration with Fibrocor under which we received an exclusive option to in-license a total of four additional novel target programs after they reached the lead optimization phase.

In February 2019, we announced a global collaboration with Evotec focused on a novel small molecule program (in preclinical development) for the treatment of fibrotic diseases of the liver and other organs. Under the terms of the agreement, we are responsible for the further development and commercialization of the program.

Our OA program

Sometimes called degenerative joint disease or degenerative arthritis, OA is the most common chronic condition of the joints. OA can affect any joint, but it occurs most often in the knees, hips, lower back and neck, the small joints of the fingers, and the bases of the thumb and big toe. In 2018, about 93 million patients were diagnosed with OA in the U.S., EU5 and Japan.[13]

In normal joints, a firm, rubbery material called cartilage covers the end of each bone. Cartilage provides a smooth, gliding surface for joint motion and acts as a cushion between the bones. In OA, the cartilage breaks down, causing pain, swelling and problems moving the joint. As OA worsens over time, bones may break down and develop growths called spurs. Bits of bone or cartilage may chip off and float around in the joint. In the body, an inflammatory process occurs and cytokines (proteins) and enzymes are formed which further damage the cartilage. In the final stages of OA, the cartilage wears away and bone rubs against bone, leading to joint damage and more pain.

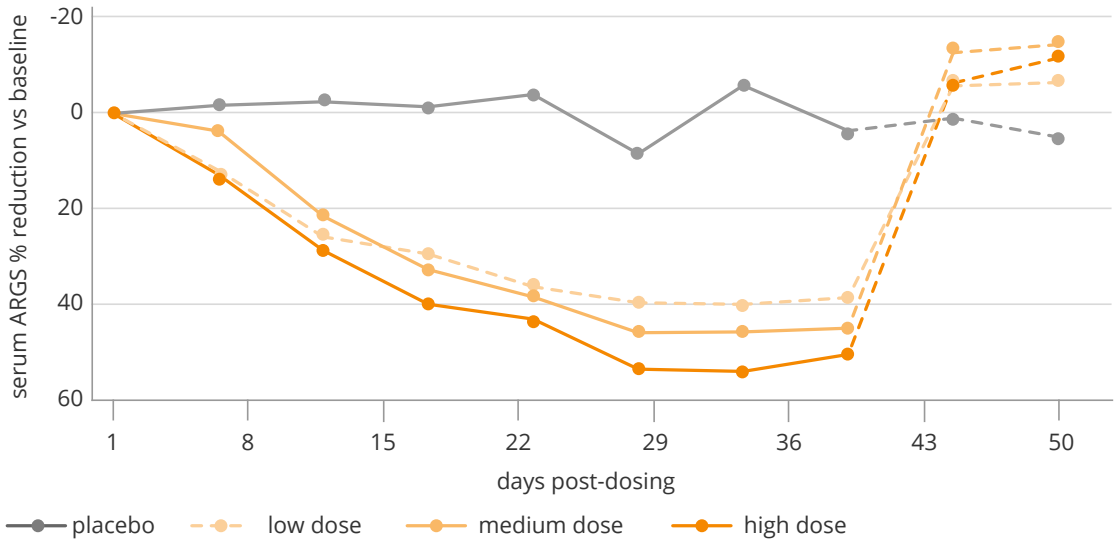
Although OA occurs in people of all ages, it is most common in people older than 65 years. Common risk factors include obesity, previous joint injury, over-use of the joint, and weak thigh muscles. One in two adults will develop symptoms of knee OA during their lives. One in four adults will develop symptoms of hip OA by the age of 85. Current treatments for OA include weight loss, physical therapy, pain and anti-inflammatory medicines, and surgery, all of which only address the symptoms of the disease. There are currently no disease-modifying therapies available for OA.

GLPG1972/S201086, also referred to as GLPG1972, is a drug candidate developed by us under our collaboration agreement with Servier. GLPG1972 acts on ADAMTS-5, a key aggrecanase involved in the breakdown of aggrecan in joint cartilage. ADAMTS-5 has been validated in the literature in both animal models and human explants, and ARGS, a byproduct of the cartilage breakdown action of ADAMTS-5, has been shown to be elevated in the joints of OA patients.

In a Phase 1b trial in OA patients in the U.S., GLPG1972 reduced the ARGS neo-epitope, a cartilage breakdown biomarker measured in the serum, by over 50% over a four-week period:

Strong reduction of ARGS

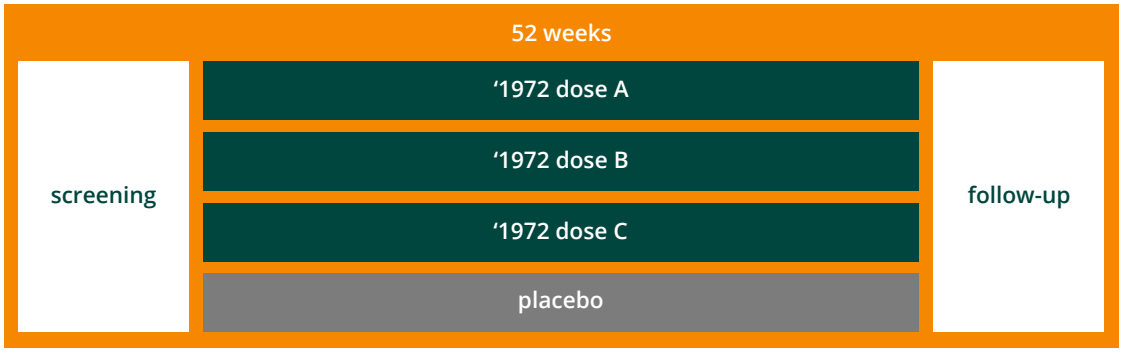
'1972 Ph1b study in OA patients



[13] Sources: Decision Resources Group, Global Data, Galapagos Custom Research

Based on these results, we and our collaboration partner Servier advanced GLPG1972 to a Phase 2b trial, ROCCELLA, the start of which was announced in June 2018.

ROCCELLA Phase 2b trial



- 850 patients with knee osteoarthritis, recruited globally
- Primary endpoint: reduction in cartilage loss at week 52
- Secondary endpoints: change in structural and clinical parameters, safety/tolerability

ROCCELLA is a multiregional, randomized, double-blind, placebo-controlled, dose ranging trial evaluating the efficacy and safety of three different once-daily oral doses of GLPG1972 in patients with knee OA. The trial is planned to recruit approximately 850 patients in up to 15 countries. We are responsible for ROCCELLA in the U.S., where we retain full commercial rights, and Servier is running the trial in all other countries.

The primary objective of ROCCELLA is to evaluate the efficacy of at least one dose of GLPG1972 compared to placebo in reducing cartilage loss after 52 weeks of treatment. Cartilage thickness will be measured using quantitative magnetic resonance imaging of the central medial tibiofemoral compartment of the target knee. Secondary objectives include safety and tolerability, several additional measures of structural progression, changes in bone area, pain, function, stiffness, and patient global assessment.

We and Servier completed recruitment of ROCCELLA in June 2019, and we expect topline data in H2 2020.

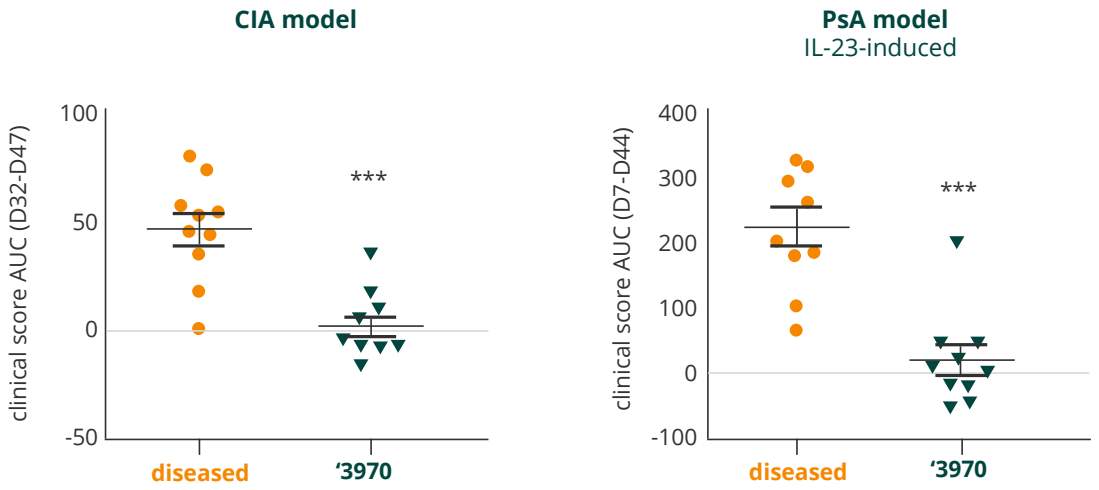
Under the terms of agreement with Servier, we are eligible to receive milestones and single-digit royalties on potential commercial sales by Servier for GLPG1972. Gilead has an option to in-license the U.S. commercial rights for GLGP1972 following completion of the ROCCELLA trial. See also further details on the collaboration with Gilead in the Notes to the consolidated financial statements.

Our Toledo program

'Toledo' is a code name for a novel target class discovered by us. Molecules inhibiting this target family effectuate a dual mode of action on inflammation by stimulating anti-inflammatory cytokines and inhibiting pro-inflammatory cytokines. We have observed unprecedented activity in various inflammatory preclinical models with compounds targeting this class.

Below are the results for Toledo compound, GLPG3970, in two preclinical models, each demonstrating a different mechanism of arthritis:

Efficacy in arthritis models with '3970



***p < 0.001

Robust efficacy demonstrated across preclinical models of arthritis

Source: internal data on file

The development strategy for Toledo is to advance multiple Toledo candidates across different selectivity profiles, and to test these in a broad panel of *in vivo* disease models targeting a number of indications.

We are now executing on a broad program to discover and develop multiple series of compounds acting on the Toledo class of targets, aimed at activity across numerous conditions, with a key focus on inflammation.

We initiated our first Phase 1 trial with GLPG3312 in early 2019 to evaluate the efficacy, safety, tolerability, and pharmacokinetics and pharmacodynamics of GLPG3312 in healthy volunteers. Later in the year we announced the start of a Phase 1 trial with the second Toledo compound, GLPG3970. We expect to launch multiple proof-of-concept patient trials in the second half of 2020 and expect to report topline data from our first patient study towards the end of the year.

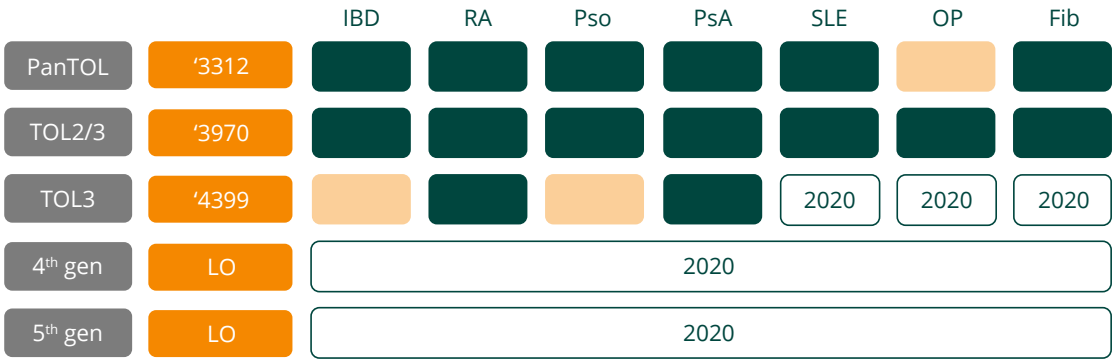
The graph below shows the current status of our Toledo program. The different disease areas that we are currently investigating are IBD, RA, psoriasis (Pso), systemic lupus erythematosus (SLE), OA, osteoporosis (OP), and fibrosis (Fib). The first generation Toledo compound, GLPG3312, has delivered promising preclinical results in IBD,

RA, Pso, PsA, SLE, and Fib. The second generation compound, GLPG3970, has shown promising preclinical results in IBD, RA, Pso, SLE, OP and Fib. The third-generation compound, GLPG4399, has shown promising results in RA and Pso, with preclinical readouts in SLE, OP, and Fib expected in the course of 2020. A fourth and fifth generation are currently in the lead optimization (LO) stage. At the time of publication of this report, it was decided to temporarily pause the start of Phase 1 studies, given de COVID-19 pandemic.

Our Toledo development strategy

- Develop multiple candidates across different profiles
- Test in broad panel of *in vivo* disease models
- Run multiple PoC trials in patients in parallel to maximize potential

Toledo: robust activity in *in vivo* models



Green: preclinical activity; orange: insufficient preclinical activity
IBD: inflammatory bowel disease; RA: rheumatoid arthritis; Pso: psoriasis; PsA: psoriatic arthritis; SLE: systemic lupus erythematosus; OP: osteoporosis; Fib: fibrosis

Gilead has an option to in-license the ex-European commercial rights to each of the Toledo molecules following completion of Phase 2 trials. See also further details in the Notes to the consolidated financial statements.

Early, deep pipeline

Beyond our Toledo programs, we continue to invest in our early stage pipeline that we built from our pool of validated targets and that we are advancing toward clinical development. Within our early stage portfolio, 15 programs are in lead optimization, five programs are evaluated in preclinical proof-of-concept studies and five are in Phase 1 development. Three molecules are part of our Toledo portfolio. In addition to targets and molecules in RA, IBD and fibrosis, we are exploring new modes of action in AS, PsA, AtD, lupus, nonalcoholic steatohepatitis, type 2 diabetes, hepatitis B, osteoarthritis, and polycystic kidney disease.

30
validated targets

15
programs in LO

5
PCCs

‘4471 – inflammation
‘4399 – inflammation
‘4259 – inflammation
‘4124 – fibrosis
‘4059 – metabolic

5
Ph1 programs

‘3312 – inflammation
‘3970 – inflammation
‘3667 – inflammation

‘555 – inflammation
‘2737 – kidney disease

Other partnered programs

MOR106

MOR106 is a human monoclonal antibody designed to selectively target IL-17C. We discovered IL-17C as a target for atopic dermatitis (AtD) and it has been shown to be distinct from other members of the IL-17 cytokine family, playing an important and pro-inflammatory role in certain skin disorders. MOR106 potently inhibits the binding of IL-17C to its receptor and thus inhibits its biological activity.

MOR106 arose from an alliance between us and MorphoSys, in which both companies contributed their core technologies and expertise and equally shared costs and benefits. In July 2018, we and MorphoSys announced that we entered into a collaboration regarding MOR106 with Novartis.

In October 2019, Novartis, MorphoSys and Galapagos jointly announced the end of the clinical development program of MOR106 in atopic dermatitis. The analysis of the program detected a low probability to meet the primary endpoint of this study. The decision was based on a lack of efficacy and not on safety concerns.

On 17 December 2019, Novartis sent us a termination notice, informing us of its decision to terminate the agreement in its entirety. The notice period for such termination is still ongoing, but we expect that such termination will become effective later this year.

CF program

Cystic fibrosis (CF) is a rare, life-threatening, genetic disease affecting the lungs and the digestive system, with 66,000 patients being diagnosed with CF in 2018 in the U.S., EU5 and Japan.[14]

Despite the approval of several drugs, there is need for better therapies to improve pulmonary function for a large majority of the patient population. Though many pediatric patients have normal lung function at the time of diagnosis, physicians generally believe that earlier treatments can have downstream benefits for the patient by slowing the deterioration in lung function.

In October 2018, we and AbbVie announced a restructuring of our CF alliance. AbbVie took over all programs in CF and will continue the development of a combination therapy for CF.

AbbVie obtained exclusive worldwide rights to the current CF drug candidate portfolio developed by the two companies in the course of the collaboration. The portfolio includes all potentiator and corrector candidates for CF, with the exception of GLPG1837 and a specific arrangement for GLPG2737. We retain rights to these two compounds for use outside the field of CF.

AbbVie is responsible for all future activities and bears all costs associated with the portfolio in CF going forward.

We are eligible to receive up to $175 million in additional milestone payments from AbbVie pending completion of certain development, regulatory, and commercial achievements in CF by AbbVie, as well as royalties ranging from the single digits to the low teens. AbbVie is eligible for future milestone payments and tiered single digit royalties on future global commercial sales of GLPG2737, if approved, in indications outside CF.

[14] Sources: Decision Resources Group, Global Data, Galapagos Custom Research