R&D

Research & Development

we **raise** the bar.

The Galapagos pipeline

We are an integrated biopharmaceutical company active in the discovery, development, and preparation for future commercialization of medicines with novel modes of action, addressing disease areas of high unmet medical need. Our pipeline comprises programs ranging from discovery to Phase 3 clinical trials in inflammation, fibrosis, osteoarthritis (OA), and other indications. Our highly flexible platform is applicable across many therapeutic areas. Our clinical stage programs include: filgotinib, which is currently in Phase 3 trials in rheumatoid arthritis (RA), Crohn's disease (CD), and ulcerative colitis (UC) and in Phase 2 trials in multiple additional indications; GLPG1690, our fully proprietary autotaxin (ATX) inhibitor, 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); GLPG1205, our fully proprietary GPR84 inhibitor which is currently in the PINTA Phase 2 proof-of-concept trial in IPF; GLPG1972, which is in the ROCCELLA global Phase 2 trial in OA patients; MOR106, which is being evaluated in Phase 1 and 2 trials in atopic dermatitis (AtD) patients; and the Toledo molecule GLPG3312, aimed at a novel class of targets discovered by us and currently in Phase 1 clinical 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, with Servier for GLPG1972, and with MorphoSys and Novartis for MOR106. In 2018 we outlicensed our CF programs to AbbVie. The following table highlights key aspects of our development program indication areas at the beginning of 2019:

Prolific late stage pipeline

area	preclinical	phase 1	phase 2	phase 3
filgotinib	10+ indications, more pivotal readouts in '19			
IPF/fibrosis	in ph3 and ph2, proprietary			
OA	ph2b underway			
AtD	ph2 underway			
inflammation/fibrosis	>20 programs			

Proprietary target discovery platform

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 cell 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 assays; and
- enables us to rapidly analyze all of the drugable genome and select pharmaceutically tractable protein targets directly by their ability to regulate key disease biology

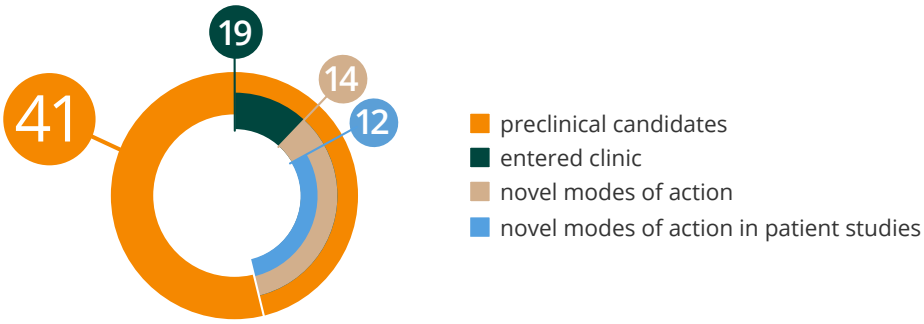
Our product candidate filgotinib acts on a target whose role in the specific disease was discovered by us using our discovery platform and we believe is a proof of success of this approach. Further proof of this approach was shown in 2017 with autotaxin inhibitor GLPG1690 in IPF patients, and with MOR106 directed toward IL-17C in AtD patients. Autotaxin and IL-17C are targets we discovered for these diseases.

The human genome is made up 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 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 drugable 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 new mode of action drugs. Since 2009, we have generated 41 preclinical candidates of which 23 have novel modes of action. Of these, 19 have entered the clinic, 12 with novel modes of action.



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, and hepatitis.

Filgotinib: selective JAK1 inhibitor with a potential best-in-class product profile

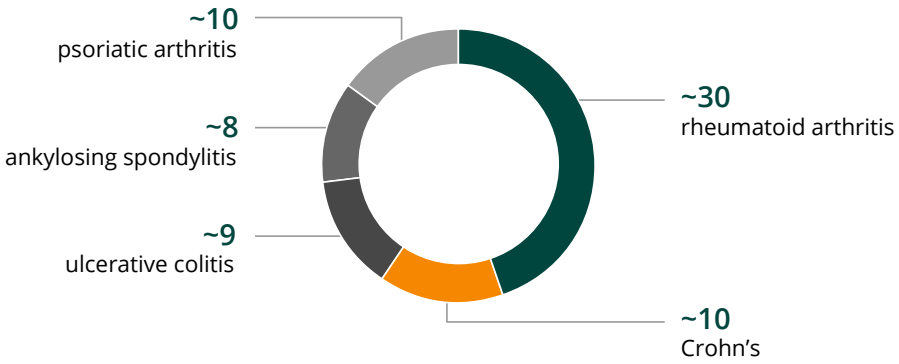
Based on results from our Phase 2 trials and the FINCH Phase 3 trials, we believe that filgotinib is a promising candidate for the treatment of RA, CD and potentially other inflammatory diseases. We are party to a collaboration agreement with Gilead to develop and commercialize filgotinib in multiple diseases. Under the terms of the collaboration, Gilead is primarily responsible for development and seeking regulatory approval of the licensed product. We are required to use commercially reasonable efforts as requested by Gilead to assist Gilead with certain development activities. Gilead initiated Phase 3 clinical programs in RA, CD, and UC in 2016, and we and Gilead initiated Phase 2 trials with filgotinib in additional indications in 2017, with the first readouts from these trials reported in 2018. The following table highlights our filgotinib program and status at the time of publication of this report:

We build a filgotinib franchise

area	phase 1	phase 2	phase 3	status
rheumatoid arthritis				recruited
Crohn's disease				recruiting
ulcerative colitis				recruited
ankylosing spondylitis				study completed
psoriatic arthritis				study completed
small bowel CD				recruiting
fistulizing CD				recruiting
Sjögren's				recruited
cutaneous lupus				recruited
lupus nephropathy				recruited
uveitis				recruiting

Markets for inflammation drugs are 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 $30 billion, with the other main markets combined representing a slightly larger opportunity than in RA:

Inflammation market in ~2027, $B



Based on the Phase 2 and 3 data observed with filgotinib in RA and Phase 2 data in CD, AS, and psoriatic arthritis (PsA) thus far, we believe that filgotinib has the potential to improve treatment standards substantially in RA, inflammatory bowel diseases (IBD), AS, and PsA. Compared with biologic agents, filgotinib is orally administered, with a rapid onset, sustained response, and potential for monotherapy. American College of Rheumatology (ACR) scores with filgotinib in Phase 2 and 3 trials in RA patients are encouraging, and CDAI remission and SES-50 scores are similarly promising with filgotinib in a Phase 2 trial in CD patients who are naïve to TNF therapy. 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 reported in all trials.

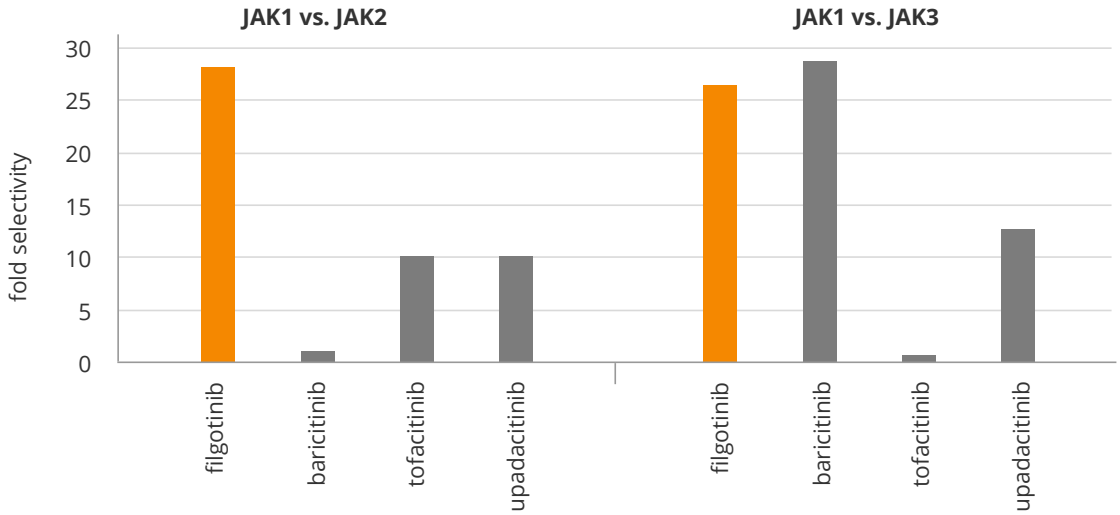
Our filgotinib program in RA

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. According to GlobalData, sales of RA therapeutics across the 10 main healthcare markets was $21.7 billion in 2017, with the current market being dominated by injectable, biological therapies. Biologics, mostly TNF therapies, often lose their effect over time, so there continues to be a considerable unmet need with regard to efficacy, safety, and convenience of use with existing treatments.

New oral therapies that target the Janus kinase (JAK) signaling pathway are emerging to treat inflammatory diseases; some JAK inhibitors, however, are associated with a range of side effects, including 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 discovered filgotinib as a JAK1 specific small molecule inhibitor. In a human whole blood assay 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.

We believe the high selectivity of filgotinib for JAK1 may allow for a positive efficacy profile, with an improved safety profile for filgotinib due to the improved selectivity over JAK2 and JAK3.

Filgotinib
High selectivity for JAK1



Source: Galapagos human whole blood assay

Source: Galapagos biochemical assay

*"*Ex Vivo* Comparison of Baricitinib, Upadacitinib, Filgotinib, and Tofacitinib for Cytokine Signaling in Human Leukocyte Subpopulations," McInnes et al, ACR 2017

DARWIN Phase 2 program with filgotinib in RA

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 and pulmonary embolisms. We believe its once-a-day oral dosage and its low risk for drug-drug interactions make it convenient for patient use.

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

DARWIN 3 is 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 filgotinib once per day or at 100 mg filgotinib twice per day (except for males in the U.S. sites of these trials who receive 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 132 weeks of treatment at ACR 2018. Promising activity levels were maintained and a favorable tolerability profile was reported. Data in DARWIN 3 were consistent with the risk/benefit profiles reported in DARWIN 1 and 2. These data were presented by Dr. Arthur Kavanaugh at the 2018 Annual Meeting of the ACR.

Based on our review of published studies, filgotinib has shown the lowest rates of infection, deep venous thrombosis (DVT) and pulmonary embolisms per 100 patient year experience (PYE) versus other JAKs and other therapy types thus far in RA:

Low incidence of DVT and infections

event per 100 PYE	filgotinib	baricitinib	tofacitinib	upadacitinib	tocilizumab	adalimumab
	50-200 mg	2 and 4 mg QD	5 mg BID	6 and 12 mg BID	4 and 8 mg/kg	
	DARWIN3 wk132	Genovese et al ACR 2017	Wollenhaupt ACR 2017	Genovese ACR 2017	Genovese ACR 2012	Burmester 2011
patient year exp.	2,042	6,637	5,278	725	14,994	23,943
serious infection	1.0	2.9	2.4	2.3	4.5	4.6
herpes zoster	1.5	3.2	3.8	3.7	ND	ND
DVT/ PE	2/2,042* 0.1	31/6,754 0.5	3/1,849 0.2	5/725 0.7	ND	ND
deaths	0.2	0.3	0.6	0.3	0.6	0.8

* one single patient experiencing DVT and PE
DVT/PE = deep venous thrombosis/pulmonary embolism
Note: data not from head-to-head studies, comparisons may not be accurate
Tofacitinib DVT/PE data from Mease, ACR 2017 (5 mg bd), and death data from 2012 FDA Medical review
Baricitinib: DVT/PE Weinblatt ACR 2017

FINCH Phase 3 program with filgotinib in RA

In August 2016, Gilead initiated the FINCH global Phase 3 program investigating the efficacy and safety of 100 mg and 200 mg filgotinib once daily, in RA patient populations, ranging from early stage to biologic-experienced patients:

FINCH 1 is an ongoing 52 week, randomized, placebo- and adalimumab-controlled trial in combination with methotrexate (MTX) enrolling 1,759 adult patients with moderately to severely active RA who have had inadequate response to MTX. The primary endpoint is ACR20 at week 12. The trial includes radiographic assessment at weeks 24 and 52. We and Gilead reported on 28 March 2019 that FINCH 1 met primary and key secondary endpoints.

FINCH 2 was a 24 week, randomized, placebo-controlled trial in 449 patients who were on conventional disease-modifying anti-rheumatic drugs (cDMARD), and had an inadequate response to biological treatment. In this study, 23.7 percent of patients had received three or more bDMARDs. The primary endpoint was ACR20 at week 12. We and Gilead reported in September 2018 that FINCH 2 met all primary and key secondary endpoints.

FINCH 3 is an ongoing 52 week, randomized trial in 1,252 MTX-naïve patients to study filgotinib in combination with MTX, as well as monotherapy. The primary endpoint is ACR20 at week 24. Radiographic progression will also be assessed. We and Gilead reported on 28 March 2019 that FINCH 3 met the primary endpoint.

In addition, Gilead is performing a dedicated male patient testicular safety trial in UC patients, called MANTA, concurrent to all Phase 3 programs. This randomized, double-blind, placebo-controlled trial is intended to enroll adult male UC patients with a treatment phase of up to 26 weeks.

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.

Top-line FINCH 1 efficacy^ 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

^ All efficacy time points assessed at Week 12 except mTSS which was assessed at Week 24

The safety profile of filgotinib in FINCH 1 is consistent with prior studies up to Week 24. Serious adverse events occurred in 4.4 percent, 5.0 percent, 4.3 percent and 4.2 percent of the patients in the filgotinib 200 mg, filgotinib 100 mg, adalimumab and placebo groups, respectively. There were five deaths, two patients were assigned to the placebo group, two to the filgotinib 200 mg group and one to the filgotinib 100 mg group. Five patients with a malignancy were also reported -- three receiving placebo, one receiving adalimumab and one receiving filgotinib 100 mg, respectively. Three venous thrombotic events were observed (two in the placebo group, one in the filgotinib 200 mg group), and there were four adjudicated major adverse cardiovascular events, two in the placebo, one in the adalimumab and one in the filgotinib 100 mg groups. The proportion of patients with herpes zoster was similar across treatment groups (filgotinib 200 mg = 0.4 percent, filgotinib 100 mg = 0.4 percent, adalimumab = 0.6 percent, placebo = 0.4 percent), as was the rate of serious infections (filgotinib 200 mg = 1.7 percent, filgotinib 100 mg = 1.7 percent, adalimumab = 2.5 percent, placebo = 0.8 percent).

FINCH 2 results

Filgotinib achieved its primary endpoint in the FINCH 2 trial in the proportion of patients achieving an 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 100mg or 200mg compared to patients receiving placebo. Topline efficacy data are summarized in the table below:

non-responder imputation	week 12			week 24		
	placebo	filgotinib 100 mg	filgotinib 200 mg	placebo	filgotinib 100 mg	filgotinib 200 mg
	(n=148)	(n=153)	(n=147)	(n=148)	(n=153)	(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

Filgotinib was generally well-tolerated in the FINCH 2 trial, with no new safety signals compared to those reported in previous trials of filgotinib. Treatment-emergent adverse events and serious adverse events were mostly mild or moderate in severity. Serious adverse events occurred in 3.4, 5.2 and 4.1 percent of the patients in the placebo, 100mg and 200mg groups, respectively. The proportion of patients who discontinued study drug due to treatment-emergent adverse events was also similar across groups. Two cases of uncomplicated herpes zoster were reported in each filgotinib group. Two MACE were identified, one subarachnoid hemorrhage in the placebo group and one myocardial ischemia in the filgotinib 100mg group. There was one case of non-serious retinal vein occlusion in the filgotinib 200mg group and no reports of VTE or pulmonary embolism. There were no deaths, malignancies, gastrointestinal perforations, or opportunistic infections, including active tuberculosis.

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).

Top-line FINCH 3 efficacy^ 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

& 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

^ Efficacy assessed at Week 24 for all endpoints

The safety profile of filgotinib in FINCH 3 is consistent with prior studies up to Week 24. Serious adverse events occurred in 4.1 percent, 2.4 percent, 4.8 percent, and 2.9 percent of patients receiving filgotinib 200 mg plus MTX, filgotinib 100 mg plus MTX, filgotinib 200 mg monotherapy and MTX alone, respectively. There was one venous thrombotic event (in the MTX group), five cases of adjudicated major adverse cardiovascular events (two in the filgotinib 200 mg plus MTX group, one in the filgotinib 200 mg group and two in the MTX group) and one malignancy (in the MTX group). There was one death, reported in the filgotinib 200 mg plus MTX group. Serious infections occurred in 1.0 percent, 1.0 percent, 1.4 percent and 1.0 percent of the patients in the filgotinib 200 mg plus MTX, filgotinib 100 mg plus MTX, filgotinib 200 mg monotherapy and MTX groups, respectively. The proportion of patients reporting herpes zoster was 0.5 percent in each of the treatment groups.

FINCH and DARWIN 3 safety

We and Gilead also announced interim safety information from four studies of the investigational compound filgotinib for the treatment of rheumatoid arthritis (RA). The data include 24 week results of the ongoing Phase 3 FINCH 1, 2, and 3 trials, and updated Week 156 safety data from the Phase 2b DARWIN 3 long term extension study in patients with RA.

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/ MTX	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

The Phase 2b DARWIN 3 long term extension trial initially enrolled 739 patients, who received filgotinib 100 mg twice daily, 100 mg or 200 mg once daily. Safety data are summarized in the table below. Results represent treatment through 156 weeks or longer, and comprise 2,203 patient-years of exposure (PYE) to filgotinib.

	number of events (events per 100 patient-years)
	PYE=2,203
serious infections	27 (1.2)
herpes zoster	34 (1.5)
DVT/PE	2 (0.1)
death	5 (0.2)
malignancy excluding NMSC	11 (0.5)
MACE	3 (0.1)

DVT, deep venous thrombosis; PE, pulmonary embolism; NMSC, non-melanoma skin cancer; MACE, major adverse cardiac events

Our filgotinib program in inflammatory bowel disease (IBD)

IBD includes CD and UC. We observed high activity and a favorable safety 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 leads us to believe the product candidate may show activity and tolerability in UC patient trials as well. IBD affects approximately two million patients (of which approximately 0.5 million are being treated with biologics) in the United States and Europe, and the market for IBD therapies is approximately $9 billion today, according to GlobalData. Current treatments are dominated by anti-TNF agents, with new biologic products gaining some ground in second line treatment.

CD is an IBD of unknown cause, resulting in chronic inflammation of the gastrointestinal (GI) tract with a relapsing and remitting course. Today, only 10% of CD patients achieve prolonged clinical remission. There are currently no highly effective oral therapies approved for CD and, similar to RA, treatment is dominated by injectable, biologic 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 we believe that filgotinib, with its high selectivity for JAK1, is a highly attractive candidate for the treatment of CD. By inhibiting JAK1 but not JAK2, unwanted effects such as anemia may be prevented. This absence of anemia is of particular importance to IBD patients, who frequently experience fecal blood loss.

Our clinical program with filgotinib in CD

Our FITZROY Phase 2 trial (174 patients) evaluated filgotinib once-daily versus placebo in patients with moderate to severely active CD and mucosal ulceration. Patients recruited were either anti-TNF naïve or anti-TNF failures. 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. As reported in *The Lancet* (Vermeire *et al*), 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%). We believe that the activity observed with filgotinib in TNF naïve patients in FITZROY compared favorably to that seen with other treatments in other, separate trials.

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.

Gilead initiated a Phase 3 trial (DIVERSITY) with filgotinib in CD in November 2016. The DIVERSITY Phase 3 trial 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 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. In the United States, males may receive 200 mg if they failed at least one anti-TNF and vedolizumab, a monoclonal anti-integrin antibody marketed by Takeda. We expect Gilead to complete recruitment for DIVERSITY in the third quarter of 2020.

In March 2017, Gilead initiated a Phase 2 trial in small bowel CD and a Phase 2 trial in fistulizing CD.

Our clinical program with filgotinib in UC

UC is an inflammatory bowel disease resulting in ulcerations and inflammation of the colon and rectum. Unlike CD, UC involves damaging inflammation of only the colon and rectum. According to GlobalData, there were 1.2 million patients being treated for ulcerative colitis in the 7 major markets, for combined total sales of just over $5 billion in 2017. 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 and could likely be achieved by a new mechanism of action.

Gilead initiated the 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 patients with moderately to severely 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 SELECTION were randomized to receive placebo, 100 mg or 200 mg filgotinib. In the United States, males may receive 200 mg if they failed at least one anti-TNF and vedolizumab.

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 proceed into Phase 3 as planned at both the 100 mg and 200 mg once daily dose level in biologic-experienced and biologic-naïve patients. Gilead completed screening for SELECTION in 2019.

Other clinical programs with filgotinib

In the course of 2017, Gilead initiated clinical trials with filgotinib in Sjögren's disease, cutaneous lupus erythematosus, lupus membranous nephropathy, and uveitis. We initiated patient trials with filgotinib in PsA and AS, for which we reported topline results in 2018. In 2019, Gilead reported completion of recruitment for Sjögrens disease and cutaneous lupus erythematosus, and that they are no longer recruiting for lupus membranous nephropathy.

Psoriatic arthritis

PsA is an inflammatory form of arthritis, affecting up to 30% of psoriasis patients. There are approximately 1 million patients in the U.S. and European Union today, with men and women being affected equally. 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.

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 moderately to severely 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%, $p<0.01$).

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).

Ankylosing spondylitis (AS)

AS, a systemic, chronic, and progressive inflammatory arthritis, is one of the most common rheumatic diseases across the globe, affecting approximately 2 million patients in the U.S., Europe, and Japan today. 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 moderately to severely 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%, $p<0.0001$).

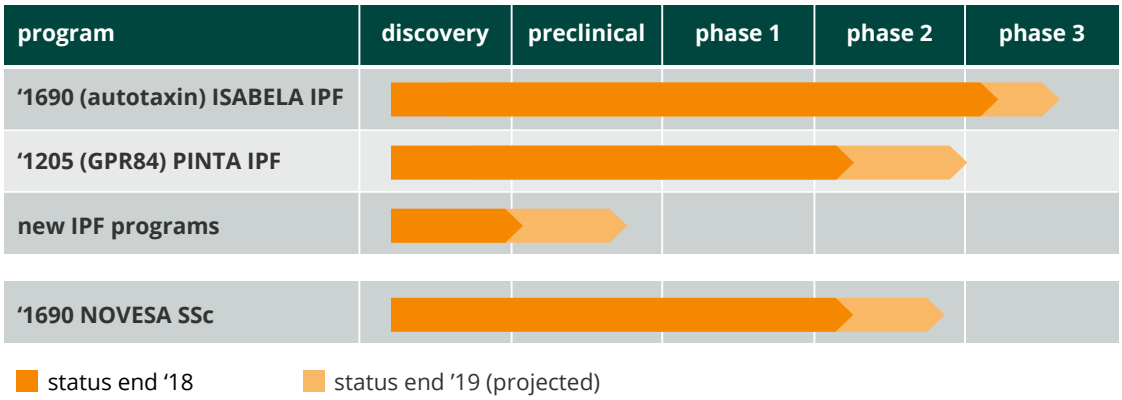
Adverse events were generally mild or moderate in severity and were reported in an equal proportion of patients in the filgotinib and placebo groups. Laboratory changes were consistent with those previously reported for filgotinib, and no new safety signals were observed in the trial. There was one treatment-emergent 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).

Our fibrosis programs

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. We also recently expanded clinical research into SSc, and plan to explore additional fibrotic indications with our earlier stage compounds in 2019.

Moreover, we actively pursue business development opportunities in the space. In January 2019, we announced a global collaboration with Fibrocor, focused on a novel target for IPF and other fibrotic indications, followed by a collaboration with Evotec for an undisclosed target in fibrosis, announced in February.

The following is an overview of our IPF portfolio and expected clinical development in 2019:



About IPF

IPF is a chronic, relentlessly progressive fibrotic disorder of the lungs that typically affects adults over the age of 40. According to GlobalData, IPF affects approximately 200,000 patients in the United States and Europe, and this population is expected to grow, in part thanks to improved diagnosis. Furthermore, prevalence is expected to increase with the aging population[3]. The clinical prognosis of patients with IPF is poor, as the median survival at diagnosis is two to four years. Currently, no medical therapies have been found to cure or stop the progression of IPF. The medical 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[4] and Ofev[5] 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 $1.9 billion in 2017, with 74% of global revenues being in the United States. 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, 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. We estimate that the market of approved IPF drugs will grow to $5 billion by 2025.

[3] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3848422/
[4] Esbriet® (pirfenidone) is an approved drug for IPF, marketed by Roche/Genentech
[5] Ofev® (nintedanib) is an approved drug for IPF, marketed by Boehringer Ingelheim

Our IPF trials

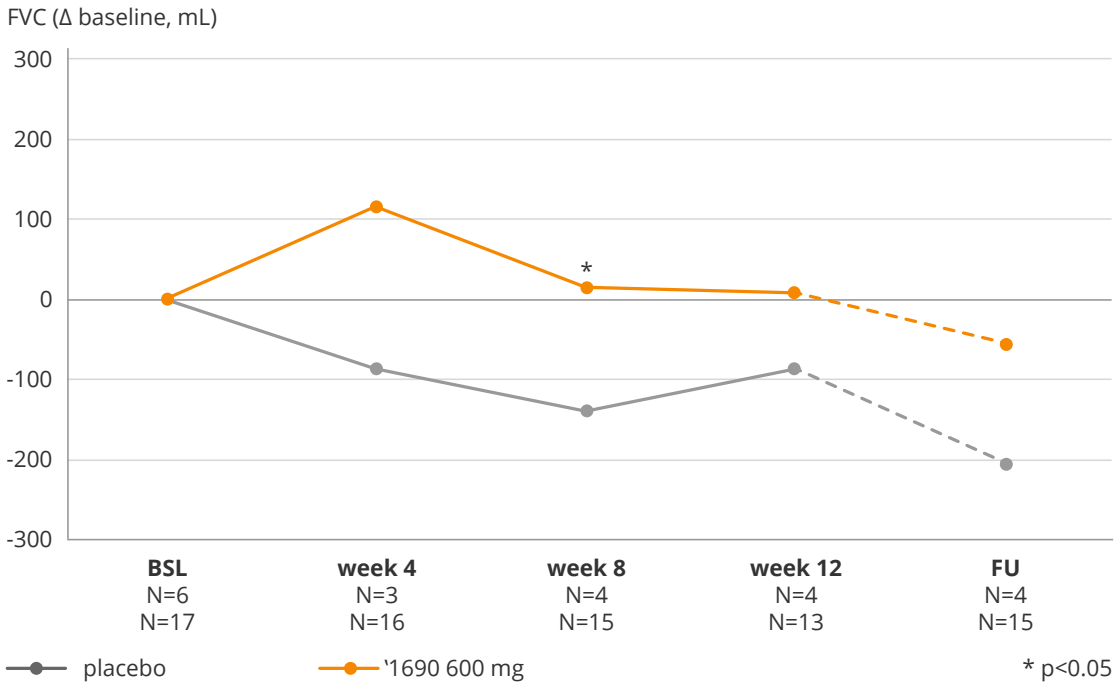
GLPG1690

Our most advanced IPF asset is our product candidate GLPG1690, a potent and selective inhibitor of ATX, which is fully proprietary to us. We identified ATX as a potential target for IPF, after finding the target using an inflammation assay in our target discovery platform. 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. Palmer *et al* published in *Chest* in 2018 on Bristol Meyers Squibb's LPA1 inhibitor tested in Phase 2, showing activity in reducing loss of Forced Vital Capacity in mL (FVC) in IPF patients. LPA1 is downstream of ATX, supporting further evaluation of ATX inhibition. 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.

In August 2017, we announced positive topline results for our Phase 2a FLORA trial in IPF patients. This randomized, double-blind, placebo-controlled trial investigated a once-daily 600 mg oral dose of GLPG1690, administered for 12 weeks in 23 IPF patients, 17 of whom received GLPG1690 and six placebo. Primary objectives of the trial were to assess 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.

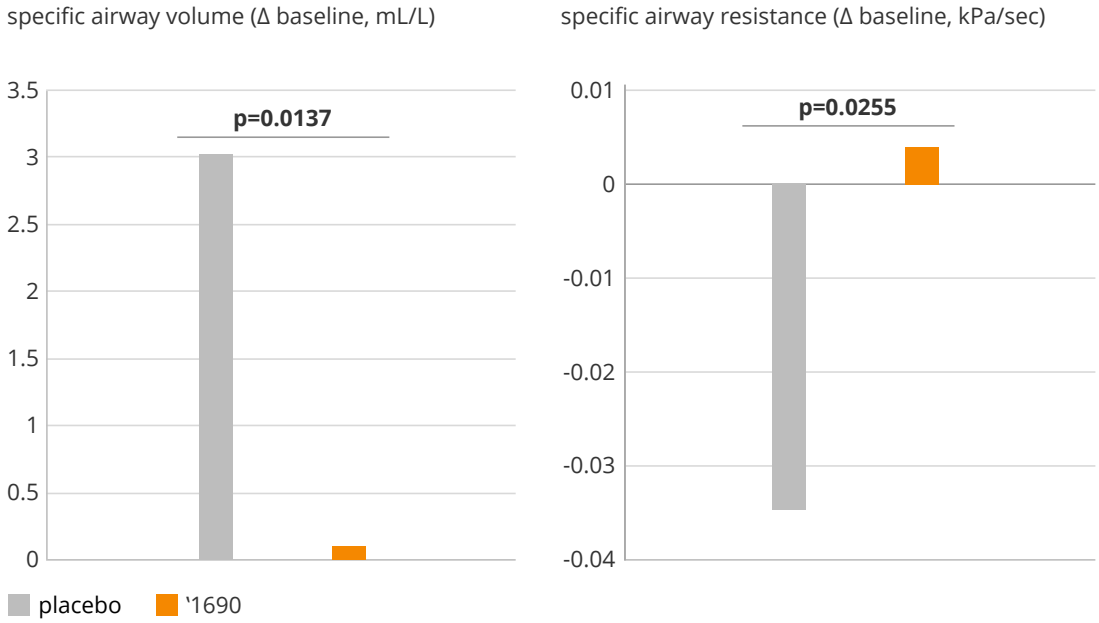
Over the 12-week period, patients receiving GLPG1690 showed an FVC increase of 8 mL, while patients on placebo showed an FVC reduction of 87 mL (mean from baseline):

FVC: stabilization by '1690



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

FRI: airway volume & resistance
Significant difference between ‘1690 & placebo



Source: Mignot et al. ATS 2018

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 trial. Rates of discontinuation due to adverse events, as well as serious adverse event rates, were similar between patients on GLPG1690 and placebo.

Following these encouraging results, 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 will continue on their standard of care and will be randomized to one of two doses of GLPG1690 or placebo. The primary endpoint will be the rate of decline of FVC (in mL) until week 52. Secondary assessments will include respiratory-related hospitalizations, mortality, quality of life, safety and tolerability.

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

- 1500 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 52 weeks
- Secondary: hospitalizations, mortality, quality of life, safety/tolerability

First patient dosing in ISABELA was announced in December 2018, and new centers are currently being opened, as recruitment efforts will continue throughout 2019.

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

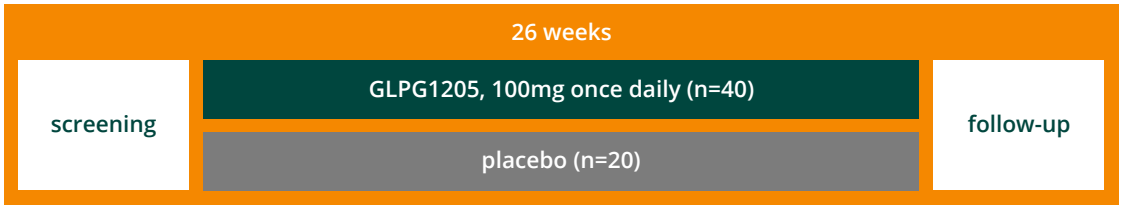
GLPG1205

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

GLPG1205 is a fully proprietary 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 100 mg 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 is planned in 10 countries in Europe, North Africa, and the Middle East. The first patient dosing was announced in October 2018, and we expect to complete recruitment of this trial in the course of 2019.

PINTA Phase 2 in IPF



- 60 IPF patients on local standard of care
- Primary endpoint: forced vital capacity (FVC) at 26 weeks
- Secondary: safety, tolerability, broad range of measurements, incl. FRI
- Recruitment in 10 countries in Europe, North Africa, & Middle East

Recruitment completion targeted Q4 '19

Note: FRI = Functional respiratory imaging

Our fibrosis trials

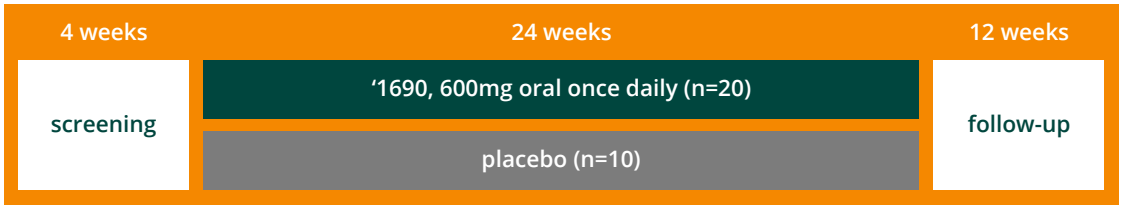
Systemic sclerosis (SSc)

SSc is a severe autoimmune disease. One of the most visible manifestations is hardening of the skin. SSc affects approximately 95,000-155,000 patients in the U.S. and Europe, with a predominance of female patients (over 75%). Broadly speaking, there are two types of SSc: limited cutaneous SSc, where the skin involvement is restricted, and diffuse cutaneous SSc. In diffuse cutaneous SSc, which represents about 35% of the SSc patient population, skin thickening affects several body areas, and patients have a higher risk of developing fibrosis of various internal organs, such as the lung.

Currently, there are no approved drugs for this disease, which has one of the highest mortality rates among rheumatic diseases. Hence, SSc represents a significant unmet medical need. Current treatment mainly consists of immunosuppressive drugs and other symptom-alleviating therapies such as methotrexate or cyclophosphamide. These aim to avoid cutaneous fibrosis, interstitial lung disease and renal crisis.

NOVESA is a double-blind, placebo-controlled Phase 2a trial evaluating the efficacy, safety and PK/PD of GLPG1690 in patients with SSc. NOVESA is planned to recruit 30 patients with diffuse cutaneous SSc.

NOVESA Phase 2 in SSc



- 30 patients with progressive diffuse (multi-organ) SSc
- Recruitment in U.S. & 5 EU countries
- Primary endpoint: modified Rodnan Skin Score at 24 weeks
- 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 24 weeks. 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.

Early in 2019 we recruited our first patient for NOVESA.

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. According to GlobalData, OA will be the fourth leading cause of disability by the year 2020. GlobalData estimates that diagnosed cases will grow to approximately 131 million cases by 2024.

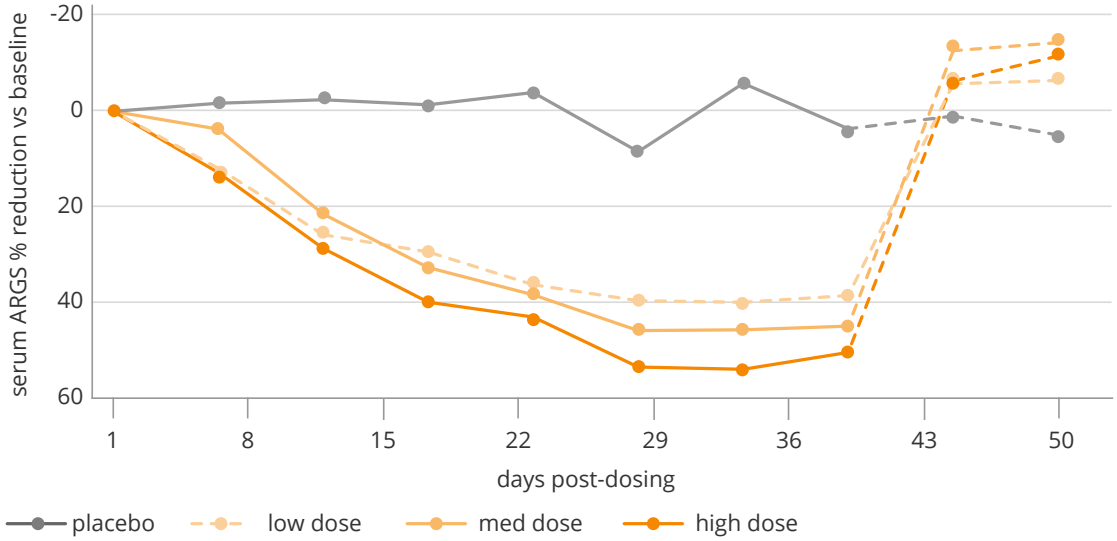
In normal joints, a firm, rubbery material called cartilage covers the end of each bone. Cartilage provides a smooth, gliding surface for joint motion and acts as a cushion between the bones. In OA, the cartilage breaks down, causing pain, swelling and problems moving the joint. As OA worsens over time, 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 develop that 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. 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 age 85. Current treatments for OA include weight loss, physical therapy, pain and anti-inflammatory medicines, and surgery, all of which address only 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 human OA patients.

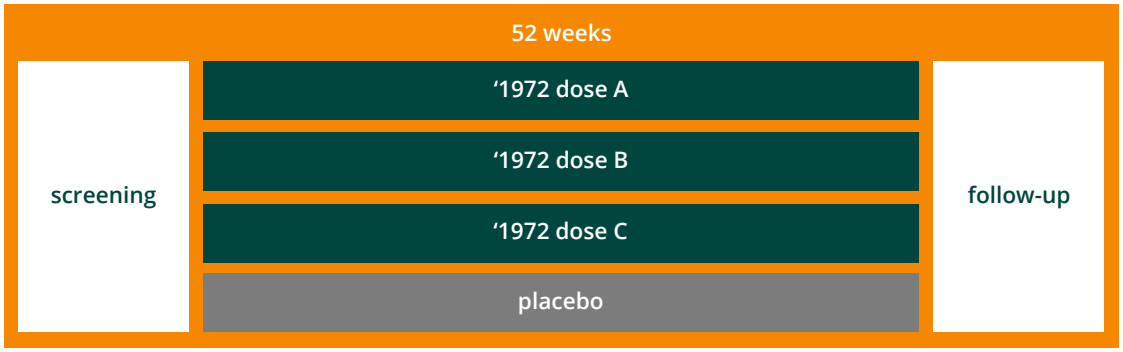
In a Phase 1b trial in OA patients in the U.S., GLPG1972 reduced the ARGS neoepitope, a cartilage breakdown biomarker measured in the serum, by over 50% over a four-week period:

Strong reduction of ARGS
'1972 Ph1b study in OA patients



Given 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 52 weeks
- Secondary: 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 osteoarthritis. 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 will run 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 intend to finalize recruitment of ROCCELLA in the second half of 2019.

We work with Servier to develop GLPG1972. We are eligible to receive milestones and single-digit royalties on potential commercial sales for GLPG1972, while we retain full commercial rights in the United States.

Our AtD program

AtD, the most severe and common type of eczema, is a chronic relapsing inflammatory skin disease that causes severe itch, dry skin and rashes, predominantly on the face, inner side of the elbows and knees, and on hands and feet. Scratching of the afflicted skin leads to a vicious cycle causing redness, swelling, cracking, scaling of the skin and an increased risk of bacterial infections. Lichenification, thickening of the skin, is characteristic in older children and adults. The National Eczema Association estimates that AtD affects over 30 million Americans or up to 25% of children and 2-3% of adults. Sixty percent of AtD patients are diagnosed in the first year of life, and 90% of patients have a disease onset before age five. Symptoms commonly fade during childhood, however, approximately 10-30% of the patients will suffer from AtD for life. A smaller percentage first develop symptoms as adults.

Generic drugs are the approved standard of care, including immunomodulators cyclosporine and mycophenolate mofetil and topical treatments. There are disease-modifying biologics and small molecules currently in development, with dupilimab (targeting IL-4Rα) most recently approved.

MOR106 is a human monoclonal antibody designed to selectively target IL-17C in clinical development worldwide. IL-17C as a target for AtD was discovered by us and 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.

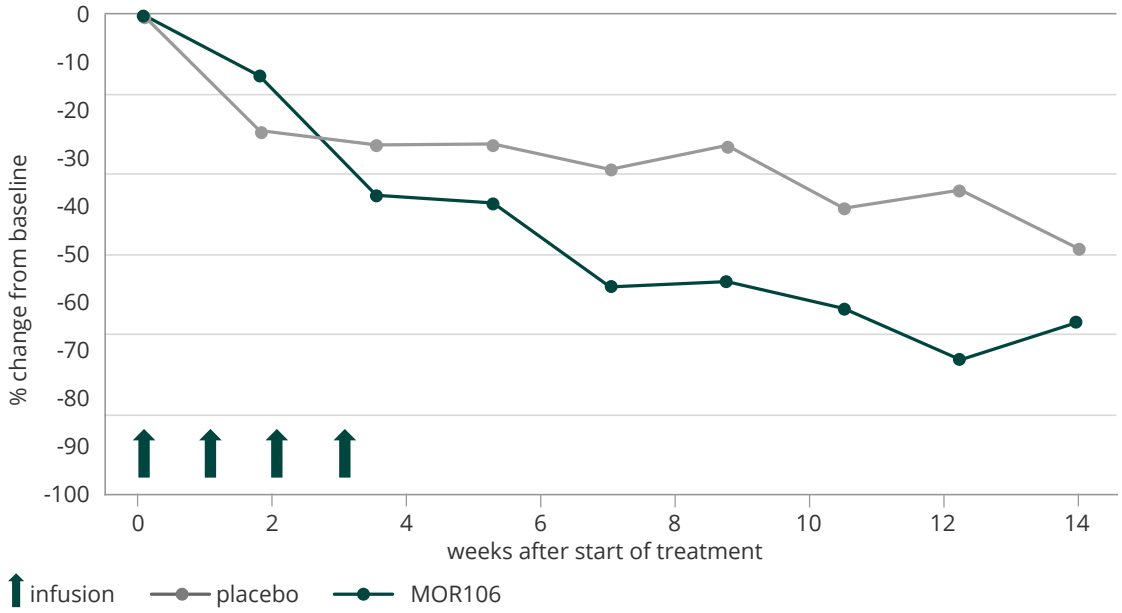
MOR106 arises from an alliance between us and MorphoSys, in which both companies contributed their core technologies and expertise and equally shared costs and benefits. In July 2018, we and MorphoSys announced that we entered into a collaboration regarding MOR106 with Novartis.

We evaluated MOR106 in a randomized, double-blind, placebo-controlled Phase 1 trial, with the first part evaluating single ascending doses (SAD) followed by multiple ascending doses (MAD) compared to placebo in approximately 25 patients with moderate to severe AtD in several European centers. Topline results of the complete trial were reported in September 2017. In the MAD portion with MOR106 in patients, all adverse drug reactions observed were mild-to-moderate and transient in nature and did not lead to clinically relevant safety signals. No serious adverse events and no infusion-related reactions were recorded.

Even though the trial was not statistically powered to show differences in efficacy between treatment groups, at the highest dose level of MOR106, in 83% of patients (five out of six) an improvement of at least 50% in signs and symptoms of AtD measured by the Eczema Area and Severity Index (EASI-50) was recorded at week four. The onset of activity was rapid and occurred within a few weeks and was maintained for over two months after the last treatment. Among patients receiving placebo, in 17% of patients (one out of six) an EASI-50 improvement was seen at week four.

As reported at AAD 2018, the pooled, mean EASI scores over time versus placebo show a sustained effect for weeks after completion of dosing:

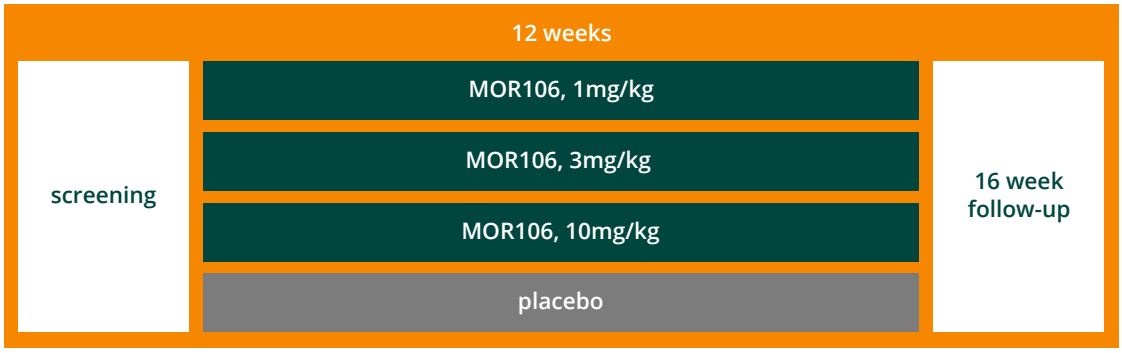
MOR106 Ph1b
EASI, % change from baseline, pooled data, median



Following the results of MOR106, together with Morphosys and then Novartis, we established a Phase 2 development program to enable a Phase 3 program that will be conducted by Novartis. We conduct all of the Phase 2 clinical research, with funding from Novartis.

We initiated the Phase 2 IGUANA trial with MOR106 in May 2018. This trial is aimed at evaluating various dosages and administration frequency. In the IGUANA Phase 2-trial, approximately 240 patients with moderate-to-severe AtD are treated over a 12-week period with one of three different intravenous doses of MOR106 (1, 3 or 10 mg/kg) or placebo using two different dosing regimens, in multiple centers across Europe. The placebo controlled, double-blind study will evaluate the efficacy, safety and pharmacokinetics of MOR106. Dosing at two or four-week intervals will be evaluated over the 12-week treatment period, followed by a 16-week observation period. The primary objective will be assessed by the percentage change from baseline in EASI score at week 12.

IGUANA Phase 2 trial

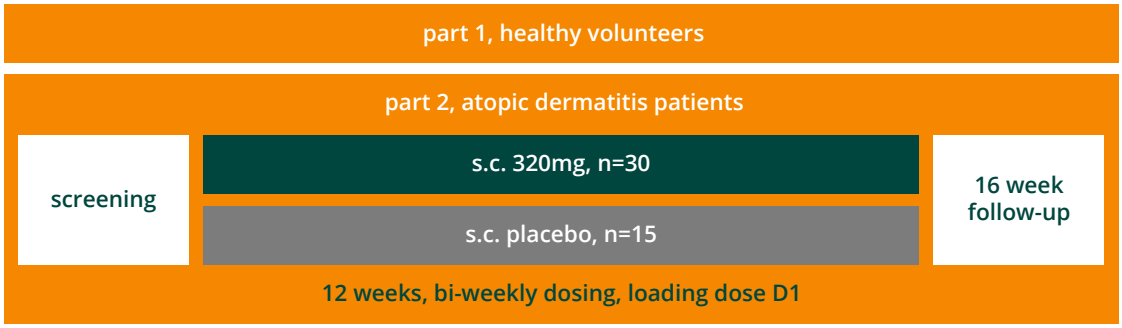


- ~240 patients with moderate-to-severe AtD
- IV infusion at 2 or 4 week intervals for 1 & 3 mg/kg
- IV infusion at 2 week interval for 10 mg/kg
- Recruitment in Europe
- Primary endpoint: % change from baseline in EASI score at week 12

We expect to report the primary analysis from IGUANA in 2019.

In September 2018 we initiated a Phase 1b bridging trial testing a subcutaneous formulation of MOR106. This bridging trial is a parallel-design Phase 1 clinical trial conducted in two parts. Part 1 is a single center, randomized, open-label trial in healthy volunteers who are treated with different single dose levels of MOR106 administered subcutaneously or intravenously. Part 2 is a multiple center, randomized, placebo-controlled, multiple dose trial in patients with moderate to severe AtD who will be treated subcutaneously for 12 weeks. Safety and tolerability, pharmacokinetics and occurrence of anti-drug-antibodies after administration of MOR106 will be assessed as endpoints. In addition, the efficacy of MOR106 will be explored in subjects with moderate to severe AtD.

MOR106 Phase 1b bridging trial



- Primary endpoints: safety, tolerability, PK
- Recruitment in EU
- Secondary endpoints Part 2: EASI/other efficacy scores, patient reported outcomes

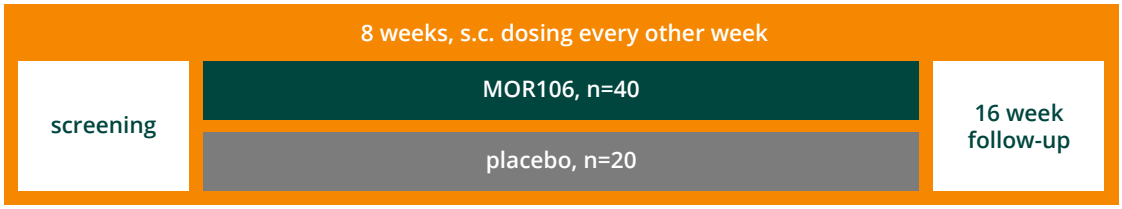
We expect to report the topline results of this trial in 2019.

We initated a Phase 2 trial testing a subcutaneous formulation of MOR106 in combination with topical corticosteroids in patients with moderate to severe AtD. This trial, called GECKO, aims to randomize 60 patients who receive either a dose of MOR106 or placebo subcutaneously for 8 weeks, together with topical steroids, with a 16 week follow-up period foreseen. The primary endpoint of GECKO is the incidence of treatment emergent adverse events and severe adverse events through day 169.

Pharmacokinetics and occurrence of anti-drug-antibodies after administration of MOR106 will be assessed as secondary endpoints. In addition, the efficacy of MOR106 will be explored.

Recruitment for GECKO will take place in the U.S. and Canada and will serve as a first trial under an IND to be submitted to the FDA.

GECKO Phase 2 trial

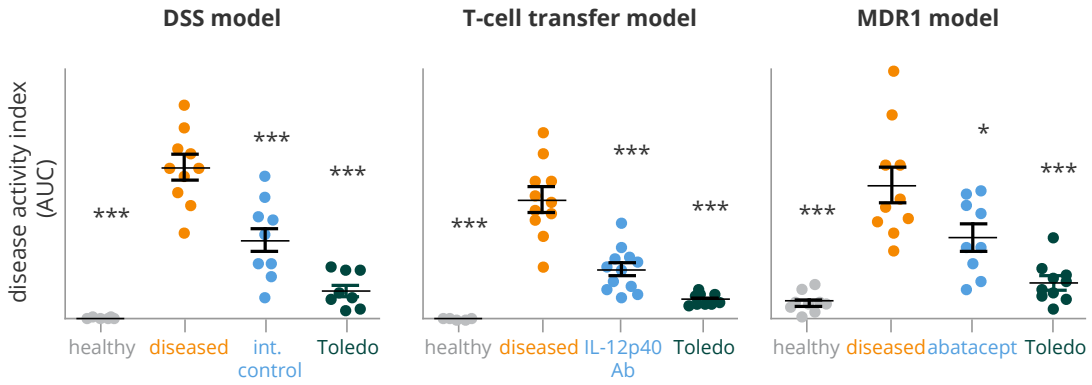


- Patients with moderate-to-severe AtD, remain on topical steroid
- Double (loading) dose on day 1 only
- Primary endpoint: incidence of TEAEs and SAEs through day 169
- Secondary measures: PK & immunogenicity
- Exploratory measures: EASI and other efficacy scores
- Recruitment in Canada & U.S.

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 the class.

Below are the results for the first Toledo compound, GLPG3312, in three preclinical models, each demonstrating a different mechanism of IBD. These results were first reported at our R&D Update in October 2018. Prior to discovering Toledo, no single compound showed activity in all three of these preclinical models in our research:



*p < 0.05; ***p < 0.001

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 up to 76 adult healthy male volunteers.

In the second half of 2019, we aim to report topline results for GLPG3312 as well as initiate a Phase 1 trial with the second Toledo compound, GLPG3970.

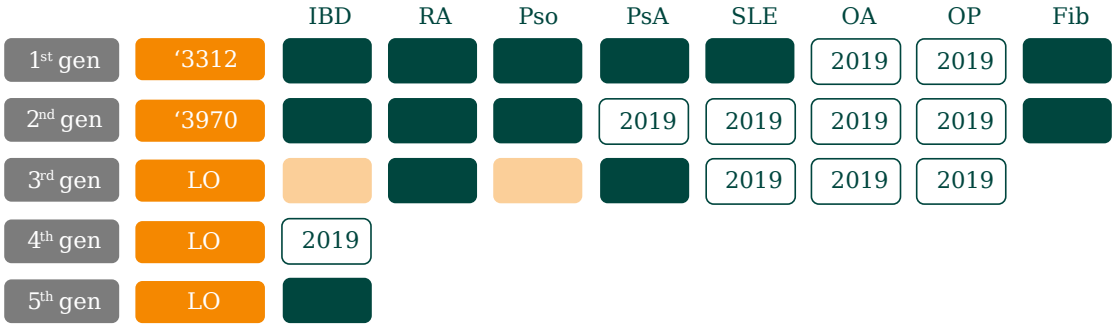
The development strategy for Toledo is to advance multiple Toledo candidates across different selectivity profiles, and to test these in a broad panel of *in vivo* disease models targeting a number of indications.

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, GLPG3312, has delivered promising preclinical results in IBD, RA, Pso, PsA and Fib, and we expect to generate preclinical data in SLE, OA and OP in 2019. The second generation, GLPG3970, has shown results in IBD, RA, Pso, SLE and fibrosis, with preclinical read-outs for PsA, SLE, OA and OP planned for 2019. The third, fourth and fifth generation are currently in the lead optimization (LO) stage.

As a next step, we plan on setting up multiple parallel-running proof-of-concept (PoC) trials in patients to investigate swiftly and efficiently the potential across the different Toledo compounds. A PoC trial for the 1st generation Toledo compound, GLPG3312, is planned for late 2019, pending satisfactory results of the Phase 1 trial currently ongoing.

Our Toledo development strategy

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



CF program

Cystic fibrosis (CF) is a rare, life-threatening, genetic disease affecting the lungs and the digestive system, impacting approximately 80,000 patients worldwide.

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 will be responsible for all future activities and will bear all costs associated with the portfolio in CF going forward.

We are eligible to receive up to $200 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.