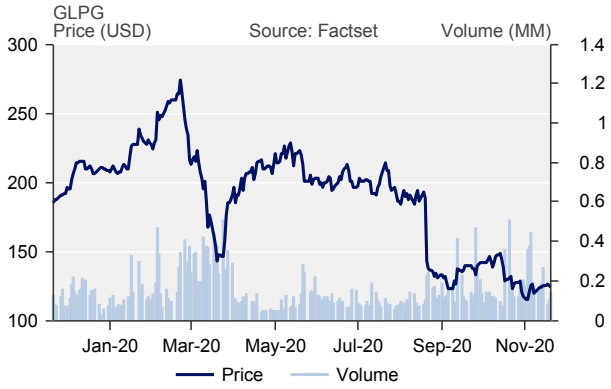
Biotechnology

GLPG - NSQ November 19, 2020

Closing Price 11/18/20	**$123.95**
Rating:	Buy
12-Month Target Price:	$170.00
52-Week Range:	$112.00 - $274.03
Market Cap (M):	8,099.0
Shares O/S (M):	65.3
Float:	99.0%
Avg. Daily Volume (000):	186.4
Debt (M):	$0.0
Dividend:	$0.00
Dividend Yield:	0.0%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Expenses ('000)

	2020E	2021E	2022E
1Q	178,771A	219,562	237,380
2Q	240,570A	226,829	247,701
3Q	208,119A	239,939	268,342
4Q	218,630	247,206	278,663
FY	846,090	933,536	1,032,086



Galapagos is based in Belgium and is listed in the US under the symbol GLPG and on the Euronext Amsterdam exchange also under the symbol GLPG. All financial data is converted to USD, from Euros.

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Galapagos NV

Buy

Pressure on Shares from CRL Creates an Attractive Entry Point – Initiating Coverage with a Buy Rating and $170 PT

Summary

- **We are initiating coverage of Galapagos NV with a Buy rating and $170 PT. Galapagos is an inflammatory and fibrotic disease company partnered with Gilead (GILD - Buy) for commercialization.**
- **Filgotinib (Jyseleca) is approved in rheumatoid arthritis (RA) in Japan and EU, but received a complete response letter (CRL) in the US. A Type A meeting is planned by YE20 to determine the path forward in RA. Filings for ulcerative colitis (UC) are expected in 1H21 in the EU and Japan, and in the US following the MANTA study (data mid-2021), around the same time as a re-filing for RA.**
- **Idiopathic pulmonary fibrosis is a potentially attractive market that Galapagos is approaching with a franchise strategy similar to Vertex (VRTX - Hold) in cystic fibrosis, developing a pipeline of potentially synergistic compounds; ziritaxestat is in P3 (futility analysis in 1H21), and GLPG1205 is in P2 (data by YE20).**
- **Conclusion. Following the CRL for filgotinib, GLPG shares have traded down to a near 52-week low, taking the market cap to less than $2B above its cash balance of $6.3B. However, little has fundamentally changed regarding the safety/efficacy profile for the company's pipeline. With key events ahead, we see significant upside in the GLPG story.**

Details

Filgotinib. Filgotinib is a second generation JAK inhibitor that is approved in the EU and Japan for RA, and has completed P3 for ulcerative colitis (UC). In the US, the compound received a CRL from the FDA, requesting the MANTA and MANTA-RAy male reproductive safety studies (data in mid-2021) and raising concerns about the risk/benefit profile of the 200mg dose. A Type A meeting is planned by YE20 to discuss the 200mg dose, which should be a significant catalyst. The 200mg dose is core to the value proposition of filgotinib, especially compared to AbbVie's (ABBV - NR) Rinvoq, which is only approved for its lower evaluated dose. We see a positive outcome as likely, considering filgotinib has a particularly clean safety profile among JAKs, and the 200mg dose appears safer than some approved JAK inhibitors. In UC, the SELECTION trials were positive for the 200mg dose (EU regulatory filing accepted). While the US filing is expected after the MANTA study, around the same time as refiling in RA.

Idiopathic pulmonary fibrosis (IPF). IPF is a progressive fibrotic lung condition that impacts 200K individuals and has a median survival of 2-5 years. Current therapies, nintedanib and pirfenidone, generated $2.8B in 2019 (up from $2.1B in 2018), despite only slowing the disease progression and having an annual discontinuation rate of 25%. Galapagos has a pipeline of potentially synergistic assets including irritates in P3 and GLPG1205 in P2, as well as compounds in early development. In P2, ziritaxestat demonstrated a stabilization of forced vital capacity decline over 12 weeks, which if confirmed in P3, should make it highly competitive. Data from the P3 is expected in 2022, with futility analysis in 1H21. GLPG1205 uses a potentially complementary mechanism and has P2 data expected in 4Q20. Galapagos' strategy in IPF is to develop multiple compounds with the potential for combination, similar to Vertex's strategy for cystic fibrosis (CF).

Attractive valuation. We model commercialization of filgotinib RA in the EU and Japan in 4Q20, and in the US in 2022 with a 50% risk adjustment, in inflammatory bowel disease (IBD) in 2022 with a 30% risk adjustment, in psoriatic arthritis (PsA) and ankylosing spondylitis (AS) in 2023, and in uveitis in 2024 with a 70% risk adjustment. We also factor ziritaxestat in 2023 with a 30% risk adjustment. A 30% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of $170.

CORPORATE PROFILE

Galápagos

Galapagos NV
Generaal De Wittelaan L11 A3 2800
Mechelen, Belgium
www.glpg.com

Investment Risk: Galapagos' has only one approved product in two regions and does not generate revenue sufficient to offset expenses.

Regulatory Risk: Galapagos' products may not be successful in clinical trials and may not meet the requirements for regulatory approval(s).

Commercial Risk: Galapagos' products may not achieve significant market share.

Financial Risk: Galapagos is not profitable and may need to raise additional capital to support operations.

Dilution Risk: Capital raises to fund operations may dilute investors.

Ownership:
Institutional: 90%
Insiders: 1%

Balance Sheet Summary:
Cash: $6.3B 3Q20A
Debt: $0 3Q20A

Analysts Covering the Stock (other than Maxim): 12

Buy: 4
Hold: 7
Sell: 1

Company Background. Galapagos (Euronext & NASDAQ: GLPG) is a clinical-stage biotechnology company specialized in the discovery and development of small-molecule medicines with novel modes of action. Our pipeline comprises Phase 1, 2, and 3 studies, pre-clinical studies, and discovery programs in inflammation, fibrosis, and other indications. The company seeks to develop a robust portfolio of clinical-stage breakthrough therapies that have the potential to revolutionize existing treatmxent paradigms. Galapagos' ambition is to become a leading global biopharmaceutical company, focused on the discovery, development, and commercialization of innovative medicines that will improve people's lives.

Galapagos' mission is to develop first-in-class medicines based on the discovery of novel targets. Using human primary cells and patient cells, the company discovers which proteins ('targets') play a key role in causing diseases such as rheumatoid arthritis, inflammatory bowel disease and fibrosis. Galapagos then aims to develop small molecules that inhibit these targets, restore the balance, and positively influence the course of the disease. This approach addresses the disease rather than just treating the symptoms. Galapagos' aim is to make a lasting positive contribution to society through the discovery of breakthrough therapies for diseases with large unmet medical need.

The Galapagos group, including fee-for-service subsidiary Fidelta, has approximately 700 employees, operating from its Mechelen, Belgium headquarters and facilities in the Netherlands, France, Switzerland, the US, and Croatia.

Senior Management:

Onno van de Stolpe - Chief Executive Officer - Onno van de Stolpe founded the company in 1999 and has served as its Chief Executive Officer and a member of the Board of Directors from 1999. From 1998 to 1999, he was the Managing Director of Genomics at IntroGene BV (later Crucell NV, which was acquired by Johnson & Johnson Services, Inc. in 2011). Prior to joining IntroGene in 1998, he was Managing Director of Molecular Probes Europe BV. He established the European headquarters after joining Molecular Probes, Inc. in the United States. Mr. van de Stolpe started his career as Manager of Business Development at MOGEN International NV in Leiden. He received an M.Sc. degree from Wageningen University. Mr. van de Stolpe has previously served as a member of the Board of Directors of DCPrime BV and as a member of the supervisory board of the Stichting Institute for Human Organ and Disease Model Technologies.

Piet Wigerinck, Ph.D. - Chief Scientific Officer - Piet Wigerinck joined in April 2008 as SVP Development and was appointed Chief Scientific Officer in 2012. Under his leadership, Galapagos has developed a large pipeline of novel mechanism of action drug candidates. He has supervised multiple successful proof-of-concept patient studies, including filgotinib, GLPG1690, and MOR106. Prior to his tenure at Galapagos, Dr. Wigerinck was Vice President, Drug Discovery, Early Development, and CM&C at Tibotec-Virco Comm VA (a subsidiary of Johnson & Johnson Services, Inc.). Under his leadership at Tibotec, TMC114 (Prezista™) and TMC435 (Olysio™) were selected and moved forward into clinical trials. Dr. Wigerinck played a key role in Tibotec's expansion into novel diseases such as Hepatitis C and advanced several compounds into Phase 1 and Phase 2 clinical trials. Dr. Wigerinck has over 30 years of R&D experience in the pharmaceutical industry and biotechnology. He holds a Ph.D. from the KU Leuven and is inventor on more than 25 patent applications. In May 2018, Dr. Wigerinck was elected as independent board member of Ipsen SA, France.

Bart Filius, MBA - Chief Operating Officer & Chief Financial Officer - Bart Filius has served as Chief Financial Officer since December 2014 and as Chief Operating Officer since September 2017. Prior to that, Mr. Filius worked over 13 years at Sanofi SA, where he was the Chief Financial Officer of Sanofi Europe during the last three years. Earlier at Sanofi, Mr. Filius was the Country Manager and Chief Financial Officer of Sanofi in the Netherlands. Before that, he was Vice President for Mergers & Acquisitions, during which time Mr. Filius led and completed the divestiture of various franchises. Prior to joining Sanofi, he was a strategy consultant at Arthur D. Little. Mr. Filius has an MBA from INSEAD and a bachelor's degree in business from Nyenrode Business University. In May 2019, Mr. Filius was appointed to the supervisory board of ProQR Therapeutics NV.

INVESTMENT SUMMARY

Bull Case. Galapagos is an inflammatory and fibrotic disease company developing a pipeline using its target discovery platform. The company is partnered with Gilead, which has the option to in-license its entire pipeline with milestones in the hundreds of millions of dollars. The partnership provides Galapagos with a robust commercial partner, as well as non-dilutive capital. The lead asset, Jyseleca (filgotinib) was recently approved in the EU and Japan for RA, and despite the complete response letter (CRL) in the US, FDA approval is likely to follow pending the MANTA and MANTA-RAy studies, which are expected to read out in 2021. The company also plans to file for ulcerative colitis (UC) in the near future, where it could be the first Janus kinase (JAK)-approved. Inflammatory disease represents a huge market, and JAK inhibitors (especially those in the second generation like filgotinib and Rinvoq) could represent an important part of the treatment paradigm going forward. In Europe, JAK inhibitors represent 20% of the market and Bulls believe that the US and Japan could follow this trend. The nearest competitor is AbbVie's Rinvoq (upadacitinib), but considering that filgotinib has one of the cleanest safety profiles among JAK inhibitors, it could have a competitive advantage in the market. Additionally, Gilead is seeking a Type A meeting with the FDA to determine whether the larger dose (200mg) could be approved in the US. This could represent a significant catalyst, if positive, as AbbVie only sought approval for one dose of Rinvoq. The inflammatory market is expected to grow significantly in coming years to as much as $65B by 2027 and while rheumatoid arthritis (RA) currently represents the bulk of the market, smaller indications (such as inflammatory bowel disease, psoriatic arthritis, and ankylosing spondylitis) are expected to grow at a faster rate, making up a larger proportion of the future market. Considering that these indications have fewer options than RA, there is blockbuster potential for filgotinib if it can capture a significant share of these markets as well. That said, filgotinib is only a component of the Galapagos story. The company is also building a pipeline in idiopathic pulmonary fibrosis (IPF). The lead asset, ziritaxestat is in P3, with a futility analysis coming up in 1H21 and its second asset, GLPG1205 is reading out P2 in 4Q20. IPF is a significant market opportunity. There are currently only two drugs approved, nintedanib and pirfenidone, which generated $2.8B in 2019 (up from $2.1B in 2018). These drugs have a 25% annual discontinuation rate and median survival for the disease remains around 2-5 years, so new drugs are needed in the market. Unlike the existing drugs, which slow the decline, ziritaxestat demonstrated disease stabilization in P2, if this is confirmed in P3, we expect the drug could capture significant market share. Galapagos also has the Toledo platform, which represents a novel inflammatory target that approaches rebalancing immunoregulatory and pro-inflammatory signals, rather than immune suppression (where a good proportion of the severe side effects for anti-inflammatory drugs come from). With multiple compounds in early development, including one in P2 for UC, RA, and a P1 from psoriasis, the Toledo platform could drive value in the long term. Galapagos shares are trading near a 52-week low following the CRL and recent fail in osteoarthritis bringing the company's market cap to ~$8B, less than $2B above cash.

Bear Case. Galapagos' valuation is hinged on three components, filgotinib, the IPF franchise, and the technology platform (including earlier-stage assets). The filgotinib (filgo) CRL in the US delays entry into the largest market for inflammatory drugs by far. With the MANTA studies reading out in mid-2021, filgo is unlikely to reach US patients until 2022. While JAK inhibitors have achieved a significant share of the inflammatory market in the EU, the same has yet to occur in the US. This is partially due to US regulatory authorities taking a more conservative stance on the class. With this in mind, Bears are more skeptical of Gilead's ability to get buy-in from the FDA for the higher dose (200mg), which is likely to determine the path forward in non-IBD indications. Additionally, the CRL was based on safety (specifically impact on sperm count, which is the point of the MANTA trials). Considering safety is the key competitive proposition for filgotinib, this is another factor that adds commercial risk. By any metric, Gilead is experienced in commercializing large drugs, but the company does not have the experience in the inflammatory disease space, while AbbVie (through Humira) is a dominant player in the space, so competition is likely to be significant. IPF is a potentially attractive market, but we won't have data for ziritaxestat until 2022. With filgotinib on hold in its largest market and the loss of OA, Bears remain more risk averse heading into the Type A meeting.

Our Take. Galapagos is an innovator in the large inflammatory and fibrotic disease market. While much of the focus for the company has been on filgotinib, it's important, in our view, to take into consideration the company's platform discovery technology, which is based on gene silencing and enables Galapagos to discover novel drug targets, expanding the long-term value proposition beyond the handful of drugs in late-stage development. Filgotinib is still a central part of the story, and despite the CRL in the US, we do see favorable odds for filgotinib's approval. The nearest catalyst is the upcoming Type A meeting in 4Q20, which is likely to determine whether the 200mg dose can proceed. In our view, this is likely as the safety profile of the 200mg compares favorably to existing approved JAK inhibitors, and the drug has received approval in both the EU and Japan. JAK inhibitors have achieved significant market share in Europe (~20%), moving into earlier lines of therapy (50% of EU JAK prescriptions come from biologic naïve patients). If this trend is observed in the US, the opportunity is significant given that the inflammatory market is expected to grow to $65B by 2027 and as the second generation of JAK inhibitors is expected to overtake earlier entrants like Xeljanz (which generated $2.2B in 2019). Additionally, IBD represents a potential high-growth area for inflammatory drugs as well. Gilead plans to file for approval in the EU and Japan in the near future for UC based on the selection data, and in the US for UC and RA in 2021 following the MANTA studies. Beyond filgotinib, IPF is an attractive opportunity for Galapagos. The company has two drugs in the clinic (ziritaxestat in P3 and GLPG1205 in P2 with data later this year) as well as three drugs in preclinical. Galapagos is approaching IPF with a similar strategy to Vertex in cystic fibrosis (CF), building out a franchise with multiple classes of medication as well as potential combinations. IPF could potentially be an even bigger opportunity than CF, with nearly 200k patients and a 25% annual discontinuation rate. Current therapies only slow the progression of the disease, while ziritaxestat appeared to stop progression in its P2 trial. Consider the median survival is currently 2-5 years, if this is confirmed in the P3, it could capture significant market share and possibly expand the already growing market. The Toledo platform also represents a long-term growth driver, with a novel approach to inflammation, rebalancing immune regulation, and pro-inflammatory signaling, rather than just suppressing the immune system. As more information and data emerges for this platform, we expect to see a compelling value proposition as immunosuppression is responsible for most of the severe side effects associated with anti-inflammatories. Galapagos shares

have traded down near 52-week lows following the CRL and the fail in OA, but with key catalysts approaching for filgotinib, IPF, and Toledo, a leading strategic partner in Gilead, and $6.3B+ on the balance sheet, we see upside to the current valuation.

Finances. Galapagos reported 3Q20 with revenue of $170M (collaborative revenue) and operating expenses of $208M, for a net loss of ($96M). The company ended the period with $6.3B in cash and projects an operating cash burn of ~$580M-$610M (€490M-€520M) for FY20.

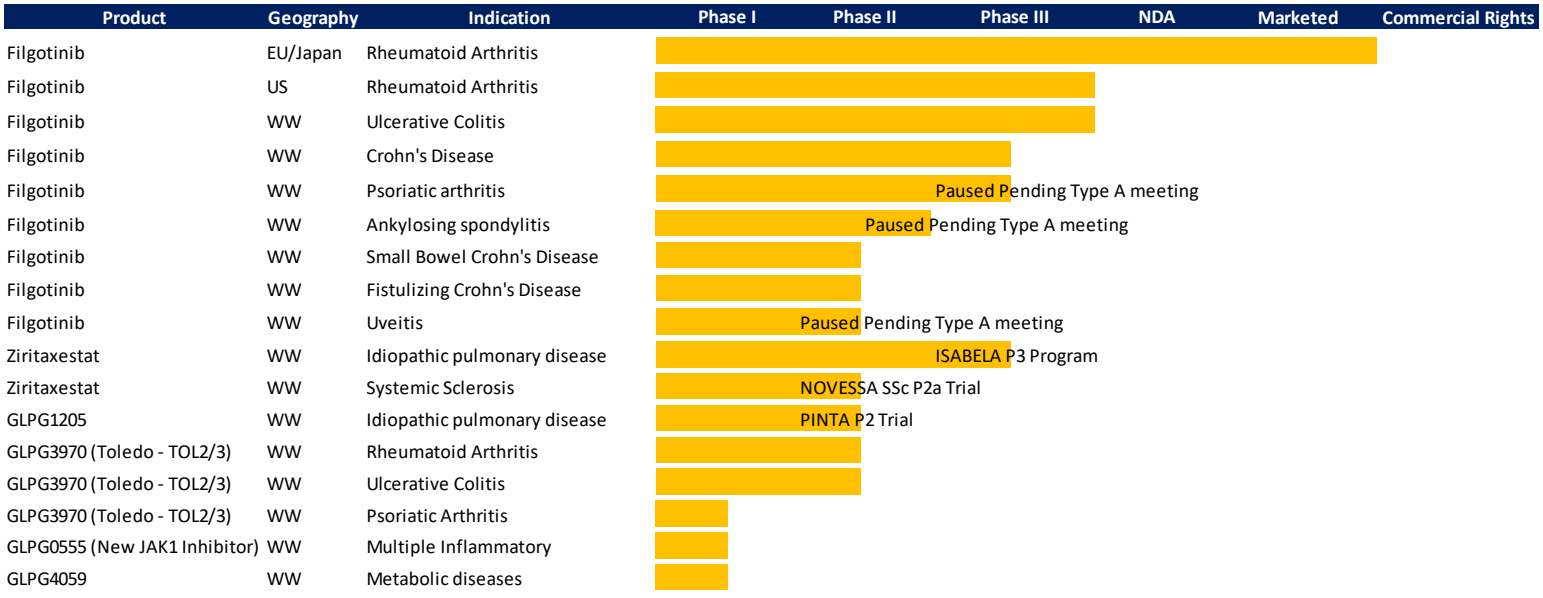
Exhibit 1. Upcoming catalysts.

Product	Geography	Indication	Event	Timeline	Impact
Jyseleca (filgotinib)	US	Rheumatoid Arthritis	Type A Meeting	4Q20	+++
GLPG1205	WW	Idiopathic Pulmonary Fibrosis	P2 PINTA Data	4Q20	++
Jyseleca (filgotinib)	Japan	Ulcerative Colitis	Filing for Approval	1Q21	+
Jyseleca (filgotinib)	US	Multiple	MANTA and MANTA-RAy Study Data	1H21	+++
Ziritaxestat	WW	Idiopathic Pulmonary Fibrosis	P3 ISABELA Futility Analysis	1H21	++
Jyseleca (filgotinib)	US	Rheumatoid Arthritis	Re-Filing NDA	Mid-2021	+
Jyseleca (filgotinib)	US	Ulcerative Colitis	Filing NDA	Mid-2021	+
Toledo	WW	Multiple Inflammatory	Proof of concept data	Mid-2021	++
Jyseleca (filgotinib)	EU	Ulcerative Colitis	EMA Approval	2H21/1H22	+++
Jyseleca (filgotinib)	Japan	Ulcerative Colitis	PMDA Approval	2H21/1H22	+++
Jyseleca (filgotinib)	US	Rheumatoid Arthritis	FDA Approval	YE21/2022	+++
Jyseleca (filgotinib)	US	Ulcerative Colitis	FDA Approval	YE21/2022	+++
Ziritaxestat	WW	Idiopathic Pulmonary Fibrosis	P3 ISABELA Topline Data	1H22	+++

Stock Significance Scale: + of moderate importance; ++ higher level; +++ very important

Source: Company reports and Maxim Forecasts

Exhibit 2. Pipeline.

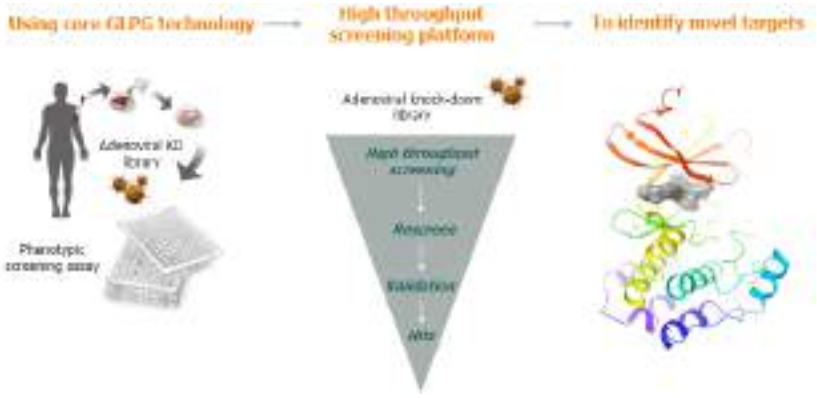


Source: Company Reports and Maxim

Galapagos Business Model

Galapagos is primarily a novel target discovery company with a proprietary discovery platform. The company uses human primary cells and patient cells to discover target proteins via gene silencing. Using engineered adenoviruses (the same technology used in gene therapy), Galapagos inserts a short sequence of DNA specific to a single gene. This new strand of DNA is translated into siRNA, which interferes with the mRNA of the protein it was designed for. Essentially, this "knocks down" production of the specific protein, mimicking what a small molecule inhibitor would do. These targets are evaluated to see if silencing that protein has a beneficial effect on the diseased cell. Galapagos now has more than 20k viruses that address ~6k druggable genes Once the target is validated, it is tested against a collection of small molecules to identify which chemical structures interact with the target to block or activate protein production. The chemical structures are then optimized for drug-like properties so that it can be moved into the clinic. This platform was used to develop filgotinib, ziritaxestat, as well as the new Toledo platform, which has a novel target.

Exhibit 3. Galapagos target discovery platform.



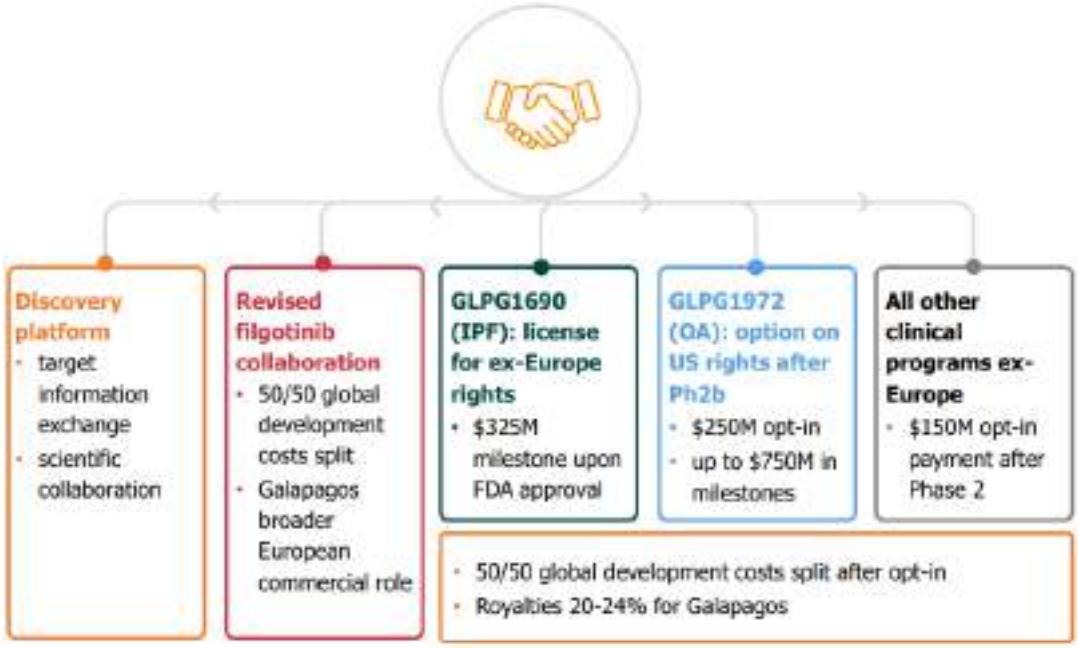
Source: Toledo Roundtable Presentation

Galapagos is also partnered with Gilead for commercialization of its products. Gilead has opt-in rights for all of Galapagos' compounds once it completes P2b with a $150M payment to Galapagos. Gilead has the commercial infrastructure and experience to handle blockbuster drugs, as well as experience building disease franchises (as is the plan for IPF) as evidenced by Gilead's dominance in HCV with the launch of Sovaldi, and subsequent launches of Harvoni and Epclusa, which generated nearly $20B at its peak in 2015, as well as HIV, which currently generated more than $16B in 2019 as its most recent entry, Biktarvy, grew market share.

Exhibit 4. Gilead collaboration financials. Galapagos and Gilead entered into a 10-year global R&D collaboration agreement in 2019. Under the deal, Gilead paid Galapagos $3.95B as an upfront payment and has purchased $1.1B of equity at €140.59 per share (a 20% premium to the 30 day VWAP). The equity purchase brought Gilead's stake in the company to 22%, from 12.3%, after which, Gilead exercised a warrant to increase its stake to ~25%, bringing the total equity purchase to $1.5B. Under the collaboration, Gilead has the right to opt-in to any of Galapagos' programs once it completes P2 for a $150M milestone (ex-filgotinib, see below), after which, the two companies will share development costs 50/50 and Gilead will receive ex-European commercial rights with a 20%-24% royalty to be paid to Galapagos. For GLPG1690 (ziritaxestat), Gilead will pay a $325M milestone on US approval. Gilead also has 2 warrants for the company, the first has a 1-year term at the same price as the equity purchase and would bring ownership to 25% (this has already been exercised), the second has a 10-year term at a 20% premium and would bring ownership to 29.9% (though Gilead cannot increase its equity stake beyond this over the deal term). Additionally, Gilead received two seats on Galapagos' Board.

Source: Galapagos Corporate Presentation

Exhibit 5. Gilead 10-year collaboration breakdown. Under the collaboration with Gilead, the companies will collaborate scientifically on the discovery/development platform, which should accelerate the discovery process. The companies have different talent pools, Gilead's scientists are largely centered in the United States, while Galapagos brings access to top European scientists (>500 scientists within Galapagos' research base). The filgotinib collaboration was also revised to give Galapagos a greater role in development and commercialization. Galapagos' share of development costs increased to 50%, from 20%. And the companies will also co-commercialize filgo in France, Germany, Italy, Spain and the United Kingdom, retaining the 50/50 profit share, and with Galapagos taking a larger commercial role. Galapagos retains exclusive rights in Belgium, the Netherlands, and Luxembourg. The remainder of the original deal remains intact with 20%-30% tiered royalties outside of aforementioned countries. Gilead was given exclusive ex-EU commercial rights for GLPG1690 in IPF with a $325M milestone owed to Galapagos on approval and a $250M option on US rights for the GLPG1972 program in osteoarthritis after the P2b, and an additional $200M if certain secondary endpoints are met. Gilead has the right to opt-in to any of Galapagos' other programs once it completes P2 for a $150M milestone (ex-filgotinib, see below), after which, the two companies will share development costs 50/50 and Gilead will receive ex-European commercial rights with a 20%-24% royalty to be paid to Galapagos.



Source: Gilead Corporate Presentation

Filgotinib – JAK1 Inhibitor

Role of cytokines in inflammatory conditions. Inflammatory diseases encompass a wide array of disorders, which are characterized by inflammation and heightened and prolonged immune response. While the inflammatory response is necessary for fighting infection and promoting healing in response to injury, a dysregulated immune response is a main driver behind a number of diseases including arthritis, psoriasis, inflammatory bowel disease, and lupus. Cytokine signaling has been found to underlie the pathogenesis of allergic, inflammatory, and autoimmune disorders. Inflammatory cytokines play a role in initiating the immune response:

TNFα has been found to play a central in multiple inflammatory diseases.[1] TNFα is part of the innate immune system and is expressed by several immune cells including macrophages, monocytes, neutrophils, T lymphocytes, and natural killer (NK) cells, and plays a role in several processes including inflammation, host defense against pathogens, and anti-tumor activity. Deregulation of TNFα production has demonstrated a pathogenic role in several auto-immune and anti-inflammatory diseases such as colitis, rheumatoid arthritis (RA), inflammatory bowel disease, and organ-related autoimmune disease.[2] Some of the most successful drugs in the treatment of inflammatory disease (such as Humira) are based on blockade of TNFα.

Interleukins are a large group of cytokines involved in various pro-inflammatory and anti-inflammatory processes. IL-6 is produced by cells in response to infection and tissue damage. It acts as a central mediator for immune response, inducing both the liver acute-phase response and optimal B- and T-cell response. IL-6 has been found to play a central role in multiple models of IBD and elevated IL-6

[1] Choy, E.H. and G.S. Panayi, Cytokine pathways and joint inflammation in rheumatoidarthritis. N Engl J Med, 2001. 344(12): p. 907-16.
[2] Manfred Kopf, Martin F. Bachmann, and Benjamin J. Marsland, "Averting inflammation by targeting the cytokine environment" Nature Reviews, Drug Discovery, vol. 9, 703-718, Sept. 2010.

levels have been observed in RA, Psoriasis, and colitis.[3] IL-1 is another cytokine playing a role in inflammation, which typically acts in concert with TNFα and IL-6. Though IL-1 targeting therapies were found to be inferior to TNF blockade, research has suggested that IL-1 may play a bigger role in the onset rather than the effector stage as the compound mediated autoimmunity by promoting dendritic cell maturation and by induction/expansion of T_H17 cells.

TNF inhibitors are among the most commercially successful drugs on the market. One of the most successful classes of medication to reach the market has been the anti-TNFα class of medication, which includes Humira, Enbrel, and Remicade. Many of the drugs targeting TNFα have become multibillion dollar blockbusters, for example, AbbVie's (ABBV - NR) Humira was the highest-selling drug of 2018 and generated $19.9B, Enbrel was also in the top 10, with sales of $7.1B in 2019, and Remicade generated $5.9B.[4] While these drugs have achieved significant commercial success in treatment of inflammatory conditions such as RA, as many as one-third of patients have an inadequate responds to anti-TNF agents, all of the current therapies can carry significant side effects, and there are relatively few examples of long-term remission after treatment cessation.[5,6] As such, there is a need for alternate pathways to treat inflammatory disease.

Janus kinase (JAK) signaling in inflammatory disease. Janus kinases (JAKs) are a relatively small group of kinases consisting of JAK1, JAK2, JAK3, and Tyrosine Kinase 2 (TYK2). As previously discussed, cytokines play a large role in inflammation. There are a number of receptor super families, including the TNF receptor family, the IL-1 receptor superfamily, the IL–17 receptor superfamily, the transforming growth factor (TGF) receptor superfamily, the receptor tyrosine kinase superfamily, and the seven transmembrane receptor superfamily. While these families play a large role in the pathogenesis of inflammatory diseases, they do not depend on JAK signal transduction. JAK inhibitors, a relatively new class of anti-inflammatory medication, focus on Type I and Type II cytokine receptors, a family of receptors that are used by more than 50 cytokines, interferons, interleukins, and other signaling molecules. JAKs bind directly to type I/II cytokine receptor.

JAK-dependent cytokines are a major contributor to immune-related diseases and a large body of evidence has been developed that shows blocking those cytokines can produce a therapeutic benefit. To provide some examples, IL-6 is overexpressed in multiple inflammatory disorders and antibodies targeting IL-6 have demonstrated some efficacy in rheumatological disorders, confirming its role in the pathogenesis.[7] Existing data also supports the roles of IL-12 and IL-23 in inflammatory bowel disease and psoriasis, supported by the data on ustekinumab (targets the p40 subunit of both IL-12 and IL-23). In allergic diseases (particularly asthma and eczema), IL-4, IL-5, and IL-13 have been found to play a role and antibodies have been successfully commercialized based on those targets (i.e., dupilumab for IL-4, mepolizumab for IL-5, and lebrikizumab for IL-13).[8,9,10] In addition, to the previously mentioned JAK-dependent cytokines, several others have been implicated across various inflammatory conditions including: interferons, IL-15, IL-21, granulocyte colony stimulating factor (GCSF), and granulocyte-macrophage (GM)-CSF.[11]

JAK inhibitors have emerged as a new class of anti-inflammatory drugs. Since all type I/II cytokine receptors are dependent on JAK for signaling, JAK inhibitors (JAKinibs) have emerged as a new class of anti-inflammatory drug. Despite the application to inflammatory conditions, the first JAK inhibitor to receive FDA approval (Jakafi, or ruxolitinib, in 2011) was for neoplastic conditions, rather than inflammatory as a V617 mutation in JAK2 is associated with diseases such as myelofibrosis (MF), polycythemia vera (PV), and essential thrombocythemia (ET) occurring in nearly 100% of PV patients and over 75% of ET patients.[12] Moving into the inflammatory space, however, presented additional challenges for JAK inhibitors namely regarding long-term safety. Cancer is a life-threatening disease and treatment with kinase inhibitors has been found to be safe and effective in the oncology setting. However when looking at long-term treatment in chronic-inflammatory conditions, there is a greater need for a clean safety profile and minimal side effects.

Exhibit 6. JAK inhibition pathways. Several approaches have been investigated for JAK inhibition in inflammatory diseases to including targeting JAK3, TYK2, JAK1, and Pan-JAK inhibition, each of which targets a certain set of receptors and therefore, inhibits a different group of cytokines. Many first generation JAKinibs were nonselective, as safety had already been proven safe and blockade of multiple JAKs may

[3] Ibid.
[4] Alex Philippidis, "Top 15 Best-Selling Drugs of 2018." *Genetic Engineering & Biotechnology News.* April 2019 p. 16-17.
[5] Gottenberg J, Brocq O, Perdriger A, et al. Non–TNF-Targeted Biologic vs a Second Anti-TNF Drug to Treat Rheumatoid Arthritis in Patients With Insufficient Response to a First Anti-TNF Drug: A Randomized Clinical Trial. JAMA. 2016;316(11):1172–1180.
[6] Singh JA et al. Biologics or tofacitinib for rheumatoid arthritis in incomplete responders to methotrexate or other traditional disease-modifying anti-rheumatic drugs: a systematic review and network meta-analysis. Cochrane Database Syst Rev (2016)
[7] Calabrese LH & Rose-John S IL-6 biology: implications for clinical targeting in rheumatic disease. Nat Rev Rheumatol 10, 720–727, doi:10.1038/nrrheum.2014.127 (2014)
[8] Ortega HG et al. Mepolizumab treatment in patients with severe eosinophilic asthma. The New England journal of medicine 371, 1198–1207, doi:10.1056/NEJMoa1403290 (2014)
[9] Hanania NA et al. Efficacy and safety of lebrikizumab in patients with uncontrolled asthma (LAVOLTA I and LAVOLTA II): replicate, phase 3, randomised, double-blind, placebo-controlled trials. The Lancet Respiratory Medicine 4, 781–796, doi:10.1016/s2213-2600(16)30265-x (2016).
[10] Wenzel S et al. Dupilumab efficacy and safety in adults with uncontrolled persistent asthma despite use of medium-to-high-dose inhaled corticosteroids plus a long-acting β2 agonist: a randomized double-blind placebo-controlled pivotal phase 2b dose-ranging trial. The Lancet 388, 31–44, doi: 10.1016/s0140-6736(16)30307-5 (2016).
[11] Schwartz DM, Bonelli M, Gadina M & O'Shea JJ Type I/II cytokines, JAKs, and new strategies for treating autoimmune diseases. Nat Rev Rheumatol 12, 25–36, doi:10.1038/nrrheum.2015.167 (2016)
[12] Lippert E et al. The JAK2-V617F mutation is frequently present at diagnosis in patients with essential thrombocythemia and polycythemia vera. Blood 108, 1865–1867, doi:10.1182/blood-2006-01-013540 (2006).

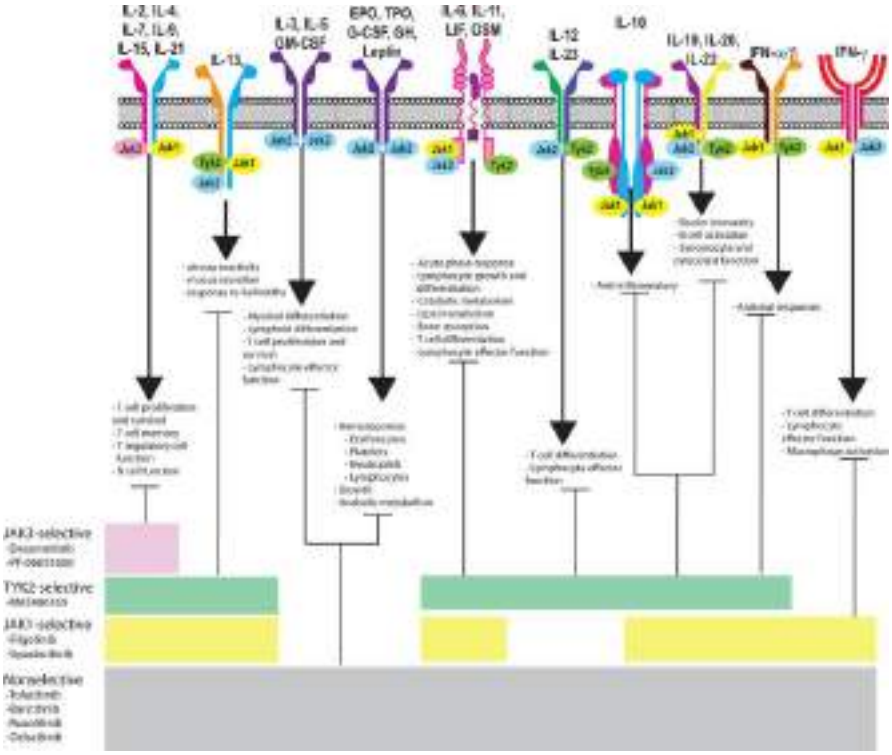
increase therapeutic efficacy. However, to minimize the prevalence of AEs (especially those arising from JAK2 inhibition such as leukopenia/anemia, Leydig cell hyperplasia, and adenoma), newer JAKinibs have been developed that selectively target certain JAK receptors.[13]

JAK1 inhibition. Targeting JAK1 has emerged as a recent strategy for inflammatory disease with the approval of AbbVie's (ABBV - NR) upadacitinib in RA, and the submission of Galapagos' filgotinib for approval in RA. JAK1 is important for signaling of IFNα/β, IFNγ, IL-2, IL-4, IL-7, IL-9, IL-21, IL-6 family cytokines, and IL-10 family cytokines.

JAK2 inhibition. JAK2 selective inhibition has been evaluated in the treatment of neoplastic disorders, rather than inflammatory (i.e., pacritinib for myelofibrosis, though it was placed on clinical hold due to concerns around mortality rates). Though selective JAK2 inhibitors have seen limited clinical success, JAK1/JAK2 inhibitors have received FDA approval in both neoplastic disorders (Jakafi) and in anti-inflammatory (Xeljanz). Though JAK2 has been found to play a role in inflammatory conditions such as RA, it is thought that inhibition of JAK2 may lead to anemia and thrombopenia by interfering with erythropoietin, thrombopoietin, and GM-CSF, and that the added benefit may be outweighed by the side effects. JAK2 is important for signaling of IFNγ, IL-3, IL-5, GM-CSF, EPO, TPO, G-CSF, GH, and leptin.

JAK3 inhibition. The JAK3 inhibitors have been developed to preserve JAK1 and JAK2 signaling, and thus, in theory, remove non-immunologic adverse events. This is because JAK3 transmits signals via γ-chain associated cytokines, which primarily affect immune cells.[14] JAK2 is important for signaling of IL-2, IL-4, IL-7, IL-15, and IL-21.

TYK2 inhibition. TYK2 inhibitors have been developed to block signaling of IL-6, IL-12, and IL-23, which are implicated in several autoimmune diseases such as psoriasis, lupus, and inflammatory bowel disease. The most advanced TYK2 inhibitor is Bristol's (BMY - NR) BMS-986165, which is in Phase 3. JTYK2 is important for signaling of IFNα/β, IFNγ, IL-12, and IL-23.



Source: Schwartz, Daniella M et al. Nature reviews. Drug discovery vol. 17,1 (2017): 78.

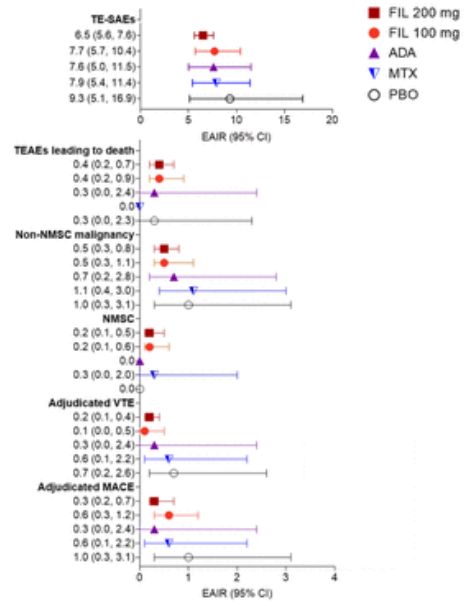
[13] Schwartz DM, et al. "JAK inhibition as a therapeutic strategy for immune and inflammatory diseases." Nat Rev Drug Discov. 2017 December 28; 17(1): 78.
[14] Macchi P et al. Mutations of Jak-3 gene in patients with autosomal severe combined immune deficiency (SCID). Nature 377, 65–68, doi:10.1038/377065a0 (1995)

Filgotinib (filgo) is Galapagos' lead candidate and is a potentially best-in-class selective JAK1 inhibitor. The compound was approved in Japan and the EU for rheumatoid arthritis in September 2020 and is marketed under the name Jyseleca. In the US, filgotinib received a complete response letter (CRL) citing concerns surrounding the risk/benefit profile of the 200mg dose and asking for the data from the MANTA and MANTA-RAy male reproductive safety studies, which read out in 2021. Filgotinib is currently under development for a number of inflammatory indications such as inflammatory bowel disease, psoriatic arthritis, ankylosing spondylitis, and lupus. The drug was discovered using Galapagos' target and drug discovery platform and has demonstrated positive results in terms of onset of action, efficacy, safety, and tolerability. The compound is partnered to Gilead, which initially paid ~$725M ($300 upfront and $425M equity investment) in December 2015. Currently, after a number of amendments to the deal structure, Galapagos is responsible for 50% of development costs (increased from 20% in the initial deal) and shares commercial rights with Gilead in France, Germany, Italy, Spain, and the United Kingdom, and will receive a 20% royalty in other regions.

Safety could be a competitive edge for filgotinib. Filgotinib is likely to enter a fairly competitive JAKinib space as the fourth approved drug in the class (in RA). Pfizer's Xeljanz and Lilly's Olumiant both carry black box warnings citing serious infections, mortality (Xeljanz only), malignancy, and thrombosis. Xeljanz had to reduce the dose in the post-marketing study, eliminating the 10mg BID arm due to an increased mortality and a statistically significant increase in pulmonary embolism noted by the Data and Safety Monitoring Board (DSMB). Upadacitinib, likely the closest competitor to filgo (as another JAK1 inhibitor), was unable to avoid a black box warning, which cites risk of serious infection, malignancy, and thrombosis. Though some investors see this as a reason for concern (if upadacitinib has a black box, filgo is likely to get one too), looking at the relative safety profiles, filgo may have an advantage based on the comparative safety profiles. The other consideration is the results of the MANTA and MANTA-RAy safety studies, which the FDA is requiring to determine the impact of filgotinib on sperm parameters in males. Both studies are fully enrolled and are expected to read out in 1H21. It is worth noting that this is a reversal from the FDA, which initially required the studies for approval, then told Gilead to proceed with filing without them, and then requested the data in the CRL, and that the EMA and Japanese regulatory authorities have not shared these concerns and have both approved filgotinib.

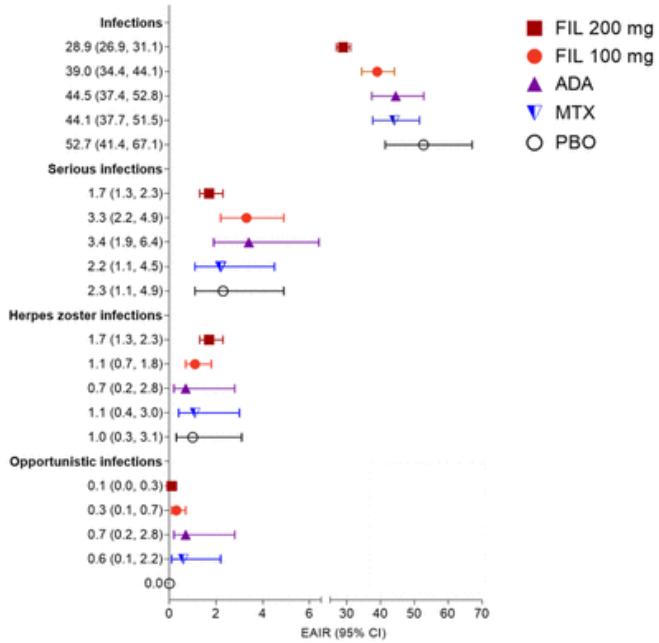
Exhibit 7. Filgotinib pooled safety analysis. A pooled safety analysis from the RA program was presented at the European League Against Rheumatism (EULAR, abstract LINK). The analysis included N=4,057 patients (n=2,227 on 200mg and n=1,600 on 100mg) across 7 clinical trials for a total of 5,493 total patient years (PY), including 3,079.2 PY for 200mg and 1,465.3 PY for 100mg. The analysis found that exposure-adjusted incidence rates (EAIRs) of serious adverse events (AEs) and treatment-emergent AEs leading to death were comparable between filgo, Humira, placebo, and methotrexate (MTX), with no dose-dependent effect. EAIR for herpes zoster infection were low overall, but slightly higher vs. placebo and Humira, and similar to MTX. Serious infection rates were comparable between filgo 100mg and Humira, and slightly lower for filgo 200mg and MTX. Rates of opportunistic infection were comparable to placebo and numerically lower than MTX and Humira. Rates of major adverse cardiac events and venous thromboembolism (VTE) were numerically lower for filgo relative to placebo.[15]

Figure 1. Rates of TE-SAEs, deaths, malignancies, VTE, and MACE by treatment group per 100 patient-years



ADA, adalimumab; CI, confidence interval; EAIR, exposure-adjusted incidence rate; FIL, filgotinib; MACE, major adverse cardiovascular event; MTX, methotrexate; NMSC, nonmelanoma skin cancer; PBO, placebo; TEAE, treatment emergent adverse event; TE-SAE, treatment-emergent severe adverse event; VTE, venous thromboembolism.

Figure 2. Rates of infections by treatment group per 100 patient-years

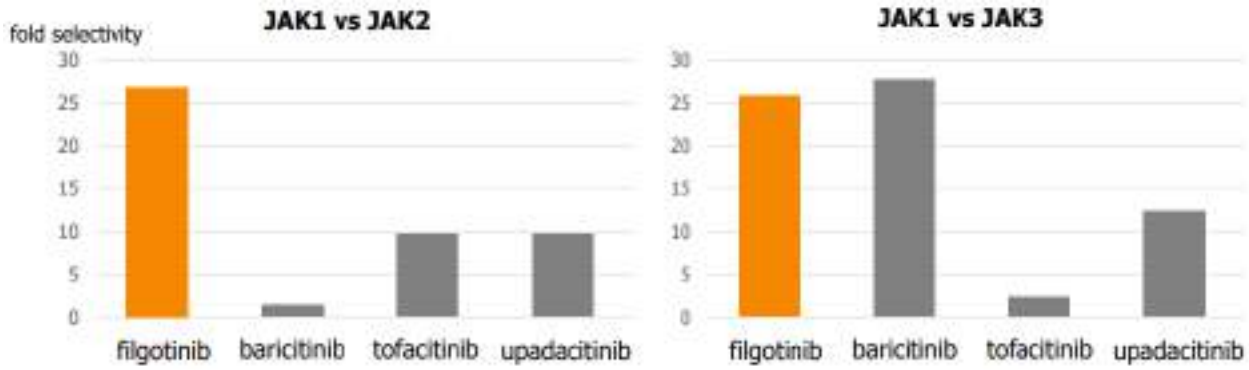


ADA, adalimumab; CI, confidence interval; EAIR, exposure-adjusted incidence rate; FIL, filgotinib; MTX, methotrexate; PBO, placebo.

Source: EULAR Abstract

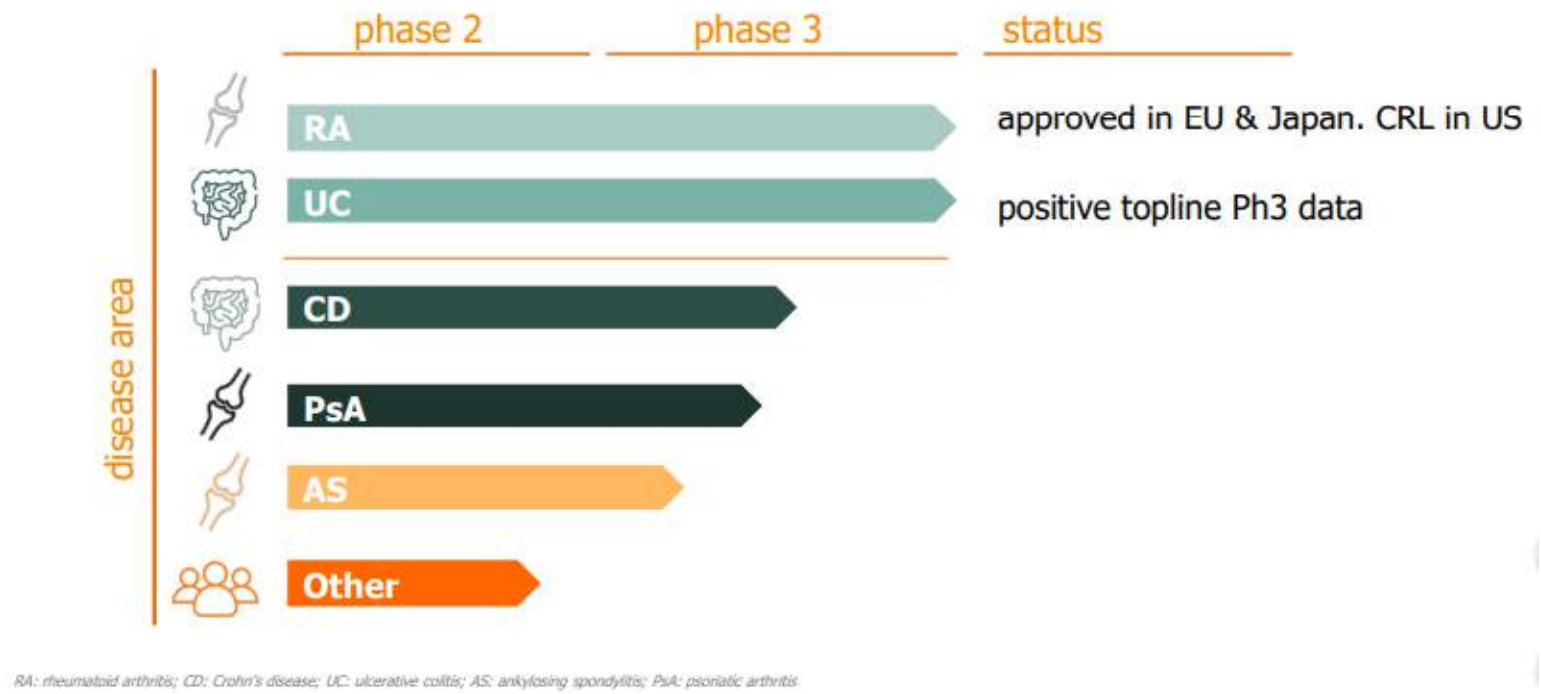
[15] Genovese MC, Winthrop K, Tanaka Y, et al. "THU0202 INTEGRATED SAFETY ANALYSIS OF FILGOTINIB TREATMENT FOR RHEUMATOID ARTHRITIS FROM 7 CLINICAL TRIALS" Annals of the Rheumatic Diseases 2020;79:324-325.

Exhibit 8. Filgotinib has best-in-class selectivity for JAK1. One big advantage for filgotinib is its best-in-class selectivity for JAK1 over JAK2, which may contribute to its strong safety profile. By comparison, upadacitinib (which also primarily targets JAK1) may also produce JAK2 inhibition at the doses required for anti-inflammatory treatment as evidenced by dose dependent decreases in hemoglobin.[16]



Source: Galapagos Corporate Presentation

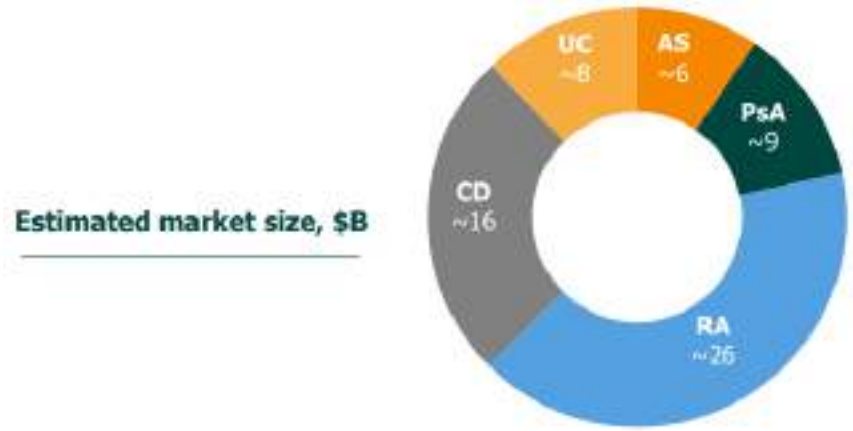
Exhibit 9. Filgotinib ongoing programs. Though rheumatoid arthritis is the lead program, filgotinib has the potential to be a broadly applicable anti-inflammatory similar to bDMARDs like Humira or Enbrel, which are available on the market. It is also worth noting that Galapagos has started a Phase 1b trial for its second JAK1 inhibitor, GLPG0555.



Source: Galapagos Corporate Presentation

[16] Genovese MC et al. Efficacy and Safety of ABT-494, a Selective JAK-1 Inhibitor, in a Phase IIb Study in Patients With Rheumatoid Arthritis and an Inadequate Response to Methotrexate. Arthritis Rheumatol 68, 2857–2866

Exhibit 10. Inflammation market is expected to reach $65B by 2027. A major reason filgotinib (and many other anti-inflammatory treatments) is initially targeting rheumatoid arthritis is the size of the market. By 2027, the RA market alone is expected to be worth nearly $30B. That said, the inflammatory market is not dominated by any single indication; non-RA diseases represent ~60% of the future market, making a platform approach necessary to fully capitalize on the market growth.



Source: Galapagos Corporate Presentation

Exhibit 11. Drugs in the inflammatory space.

Drug	Company	Indications	Approval	Commercial Sales ($M)										
JAK Inhibitors				2017	2018	1Q19	2Q19	3Q19	4Q19	2019	1Q20	2Q20	3Q20	
Jakafi	Incyte (INCY - NR)	Polycythemia Vera Myelofibrosis Acute GvHD	2011	$ 1,100	$ 1,387	$ 376	$ 410	$ 433	$ 466	$ 1,684	$ 459	$ 474	$ 488	
Jakavi (EU)	Novartis (NVS - NR)	Polycythemia Vera Myelofibrosis	2011	$ 777	$ 977	$ 258	$ 284	$ 279	$ 293	$ 1,114	$ 318	$ 310	$ 335	
Xeljanz	Pfizer (PFE - NR)	Rheumatoid Arthritis Psoriatic Arthritis Ulcerative Colitis	2012	$ 1,345	$ 1,774	$ 423	$ 613	$ 599	$ 607	$ 2,242	$ 451	$ 653	$ 654	
Olumiant	Eli Lilly (LLY - NR)	Rheumatoid Arthritis	2017 (EU)	$ 46	$ 203	$ 81	$ 102	$ 115	$ 128	$ 427	$ 140	$ 145	$ 162	
Rinvoq (upadacitinib)	AbbVie (ABBV - NR)	Rheumatoid Arthritis	2019					$ 14	$ 33	$ 47	$ 82	$ 136	$ 191	
TNF Inhibitors				2017	2018	1Q19	2Q19	3Q19	4Q19	2019	1Q20	2Q20	3Q20	
Enbrel	Amgen (AMGN - NR)	Rheumatoid Arthritis Juvenile Idiopathic Arthritis Psoriatic Arthritis Ankylosing Spondylitis Plaque Psoriasis	1998	$ 5,433	$ 5,014	$ 1,151	$ 1,363	$ 1,366	$ 1,346	$ 5,226	$ 1,153	$ 1,246	$ 1,325	
Enbrel (ex-US and Canac	Pfizer (PFE - NR)			$ 2,452	$ 2,112	$ 451	$ 420	$ 415	$ 414	$ 1,699	$ 347	$ 337	$ 321	
Remicade	Jassen (J&J, JNJ - NR)	Rheumatoid Arthritis Psoriatic Arthritis Ankylosing Spondylitis Crohn's Disease Ulcerative Colitis Plaque Psoriasis	1998	$ 6,315	$ 5,326	$ 1,102	$ 1,107	$ 1,136	$ 1,035	$ 4,380	$ 990	$ 935	$ 921	
Humira	AbbVie (ABBV - NR)	Rheumatoid Arthritis Juvenile Idiopathic Arthritis Psoriatic Arthritis Ankylosing Spondylitis Crohn's Disease Ulcerative Colitis Plaque Psoriasis Hidradenitis Suppurativa Uveitis	2002	$ 18,251	$ 19,936	$ 4,446	$ 4,870	$ 4,936	$ 4,917	$ 19,169	$ 4,837	$ 4,870	$ 4,759	
Cimzia	UCB Pharma (EU: UCB - NR)	Rheumatoid Arthritis Psoriatic Arthritis Ankylosing Spondylitis Crohn's Disease Plaque Psoriasis	2008	€ 1,424	€ 1,446	€ 782		€ 930		€ 1,712	€ 842			
Simponi	Jassen (J&J, JNJ - NR)	Rheumatoid Arthritis Psoriatic Arthritis Ankylosing Spondylitis Ulcerative Colitis	2009	$ 1,833	$ 2,084	$ 524	$ 563	$ 586	$ 515	$ 2,188	$ 529	$ 546	$ 529	

Source: Maxim Research and Company Reports

Rheumatoid arthritis. Rheumatoid arthritis (RA) is an autoimmune disease in which the body's immune system—which normally protects its health by attacking foreign substances like bacteria and viruses—mistakenly attacks the joints. This creates inflammation that causes the tissue lining inside of the joints (the synovium) to thicken, resulting in swelling and pain in and around the joints. The synovium makes a fluid that lubricates joints and helps them move smoothly. If inflammation goes unchecked, it can damage cartilage, the elastic tissue that covers the ends of bones in a joint, as well as the bones themselves. Over time, the loss of cartilage can lead to reduced joint spacing between bones, causing joints to become loose, unstable, painful and lose their mobility, in some cases even becoming deformed. Joint damage cannot be reversed, and because it can occur early, doctors recommend early diagnosis and aggressive treatment to control RA.[17] About 1.5 million people in the United States have rheumatoid arthritis (RA). Nearly three times as many women have the disease as men. In women, RA most commonly begins between ages 30 and 60. In men, it often occurs later in life. Having a family member with RA increases the odds of having RA; however, the majority of people with RA have no family history of the disease.[18]

Rheumatoid arthritis market. The current global market for rheumatoid arthritis is valued around >$20B and is largely dominated by injectable biologic such as TNF inhibitors. Methotrexate (MTX) currently represents the standard of care (SoC) for numerous inflammatory disorders, and is often combined with other medications. The drug is used both as a chemotherapy and as an immunosuppressant, working through two different pathways. For inflammatory disease, a number of mechanisms are implicated, including the inhibition of enzymes involved in purine metabolism, leading to accumulation of adenosine; inhibition of T-cell activation and suppression of intercellular adhesion molecule expression by T cells; selective down-regulation of B cells; increasing CD95 sensitivity of activated T cells; and inhibition of methyltransferase activity, leading to deactivation of enzyme activity relevant to immune system function.[19] As one of the most abundant products of activated macrophages as well as activated T lymphocytes, natural killer (NK) cells, and neutrophils, TNF plays a central role in inflammatory disease by initiating and regulating the cytokine cascade during an inflammatory response. Inhibition of TNFα forms the basis of many of the biologic treatments used in RA and other inflammatory conditions), including Humira, Enbrel, and Remicade. Many of the drugs targeting TNFα have become multibillion dollar blockbusters, for example, AbbVie's (ABBV - NR) Humira was the highest-selling drug of 2019 and generated $19.1B, Enbrel was also in the top 10, with sales of $5.2B in 2019, and Remicade generated $5.9B.[20] Yet, in spite of the success of anti-TNF-therapeutics, these agents may induce severe side effects including serious infections, reactivation of tuberculosis, invasive fungal infections, and other opportunistic infections. In addition, some patients, subsequent to anti-TNF therapies for RA, have developed multiple-sclerosis (MS)-like exacerbations and demyelinating lesions. Furthermore, only 20%-50% of RA patients will respond to a given TNF therapy. Other therapies include IL-6 inhibitors like Actemra, IL-1 inhibitors like Kineret and abatacept, which binds to CD80 and CD86 (reducing T-cell activation), and rituximab, which targets B cells by binding to CD20.

Complete response letter and approval in Japan and the EU. In August 2020, Gilead received a complete response letter (CRL) from the FDA for filgotinib in the treatment of moderate to severe RA. There are two components to the CRL. The first is requesting for the data from the MANTA and MANTA-RAy male reproductive safety studies. The request for the data represents a delay to any likely potential approval until 2H21/2022 based on the expected readout of the studies in 1H21. The second reason for the CRL was concern from the FDA surrounding the risk/benefit profile of the 200mg dose for filgotinib in RA. This is a more significant potential impact as Gilead has noted that the availability of the 200mg dose is a key factor for the Jyseleca commercial strategy in RA, especially considering that AbbVie's Rinvoq was only approved for the lower 15mg dose. The Type A meeting to discuss the risk/benefit for the 200mg dose is planned for 4Q20, and represents a major catalyst as it will likely determine whether filgotinib is the path forward in RA. Gilead has halted enrollment in the ongoing psoriatic arthritis, ankylosing spondylitis, and uveitis trials pending the result of the Type A meeting. The positive result would be a clear path forward for the 200mg dose. Another outcome is that the MANTA study is required to make a judgment on the 200mg dose, and a negative result would be that the 200mg dose is not viable. That said, it is important to note that this would not apply to IBD. In our view, it is likely that the 200mg dose is approved based on the existing profile, which demonstrates 200mg has comparable safety to 100mg and has a favorable safety profile when compared to other approved JAK inhibitors.

MANTA and MANTA-RAy studies. The MANTA and MANTA-RAy studies are assessing the impact of the 200mg dose of filgotinib on sperm concentrations. These studies were previously considered the rate-limiting step for filgotinib, until an FDA meeting in mid-2019, after which, Gilead believed that it could file based on the FINCH program without the need for the MANTA data. The MANTA study is being conducted in patients with inflammatory bowel disease (N=250 estimated enrollment). Patients are randomized to filgo 200mg or placebo and receive treatment for 13 weeks, after which, semen parameters will be evaluated. Then, patients either continue on the randomized portion for 13 weeks, or switch to filgo open-label, or SoC open-label if semen parameters decrease by a predefined threshold. The primary endpoint is a 50% decrease in sperm concentration at week 13, with a secondary endpoint at 26 weeks. The MANTA-RAy study enrolls patients with rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, or non-radiographic axial spondyloarthritis (N=109 estimated enrollment). The study is designed similarly to the MANTA study, except that the randomized portion will only continue to week 13. Both studies are fully enrolled and Gilead expects data in 1H21. That said, it is not clear if the FDA will require data from longer than 26 weeks.

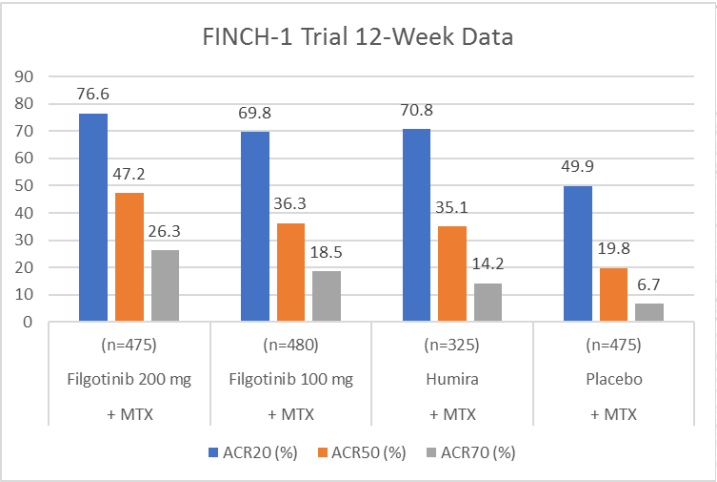
FINCH Pivotal Trials. The FINCH program consisted of three Phase 3 trials: FINCH 1, FINCH 2, and FINCH 3. The FiNCH1 trial was a 52 week trial which enrolled N=1759 patients with moderate to severely active RA and compared filgotinib in combination with methotrexate (MTX) to either placebo or Humira. FINCH 2 was a 24 week study which randomized N=449 patients who were taking a conventional DMARD but had an

[17] "What Is Rheumatoid Arthritis?" www.arthritis.org, www.arthritis.org/about-arthritis/types/rheumatoid-arthritis/what-is-rheumatoid-arthritis.php.
[18] ibid.
[19] Wessels JA, Huizinga TW, Guchelaar HJ (March 2008). "Recent insights in the pharmacological actions of methotrexate in the treatment of rheumatoid arthritis" (PDF). Rheumatology. 47 (3): 249–55.
[20] Alex Philippidis, "Top 15 Best-Selling Drugs of 2018." *Genetic Engineering & Biotechnology News.* April 2019 p. 16-17.

inadequate response to biologic therapy (23.7% of patients had tried 3 or more bDMARDS). FINCH 3 was a 52-week trial that enrolled N=1,252 MTX-naïve patients and treated them with either filgotinib or filgotinib monotherapy. The significance of the three trials in the FINCH study is that the data gathered is representative of the 3 different types of patients who would be prescribed JAKinibs: treatment-naïve patients (FINCH 3), biologic naïve patients/MTX-refractory (FINCH 1), and biologic refractory patients (FINCH 2). This breakdown of the population is demonstrated in the Olumiant patient population breakdown in Germany, where 9% of patients taking the drug are MTX-naïve, 43% are switching from biologics, and 47% are switching from a conventional DMARD (see exhibit 15). The data from the FINCH program (See exhibits 12-14) was largely positive.

DARWIN Phase 2b program. The DARWIN program consisted of three Phase 2 trials for filgotinib in RA, and was completed in 2015, with the exception of DARWIN 3, which is an ongoing long-term safety follow-up. DARWIN 1 was a 24-week dose ranging study which used three doses (50mg, 100mg, and 200mg) in twice daily and once daily schedules in combination with MTX in N=594 patients with inadequate response to MTX alone. DARWIN 2 was a similar trial that treated N=283 patients with inadequate response to MTX. The main difference was that the DARWIN 2 trial evaluated filgotinib as a monotherapy with a 4-week washout period. Both trials met their primary endpoint of ACR20 improvement at 12 weeks. The DARWIN 3 study is an ongoing long-term follow-up study that enrolled patients from both the DARWIN 1 and 2 studies after the 24 weeks were over. Female patients in DARWIN 3 were given either 200mg filgotinib per day or 100mg BID, while males were limited to 100mg per day. Patients moving into DARWIN 3 from DARWIN 1 continued to receive MTX.

Exhibit 12. FINCH-1 trial data. The FINCH-1 P3 trial enrolled N=1,729 patients with moderately to severely active RA who inadequately responded to methotrexate (MTX). Patients were randomized (3:3:2:3) to receive filgotinib 200mg (n=477), filgotinib 100mg (n=480), adalimumab (n=325), or placebo (n=477) in combination with MTX. The primary endpoint of the study was the proportion of patients who achieve an American College of Rheumatology 20% improvement response (ACR20) at week 12. Key secondaries include ACR50 and ACR70. The safety profile of filgotinib in FINCH 1 is consistent with prior studies up to Week 24. Serious adverse events occurred in 4.4%, 5.0%, 4.3%, and 4.2% of the patients in the filgotinib 200mg, filgotinib 100 mg, adalimumab, and placebo groups, respectively.



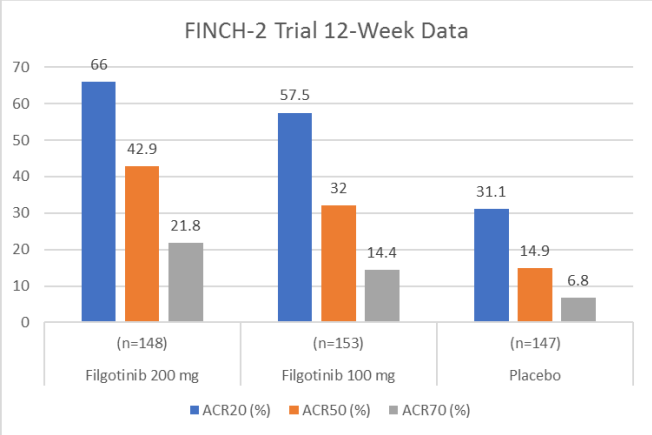
Top-line FINCH 1 efficacy* data are summarized in the table below.

	Filgotinib 200 mg +MTX (n=475)[&]	Filgotinib 100 mg +MTX (n=480)[&]	Adalimumab 40 mg +MTX (n=325)[&]	Placebo +MTX (n=475)[&]
ACR20 (%)	78.8**	69.8**	70.8	49.8
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) (%)	46.7**,†	36.0**	41.4	22.4
DAS28(CRP) < 2.6 (Clinical remission) (%)	22.9**,‡	23.8**,‡	22.7	9.3
HAQ-DI change	-0.64**	-0.56**	-0.61	-0.42
mTSS change	0.17*	0.17*	0.08	0.28

All efficacy time points assessed at Week 12 except mTSS which was assessed at Week 24
[&]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% improvement
** p <0.001, compared with placebo
† p <0.001, non-inferiority to adalimumab
‡ p <0.01, superiority to adalimumab
* Comparison not adjusted for multiplicity

Source: Gilead and Maxim Research

Exhibit 13. FINCH-2 trial data chart. FINCH 2 was a global, 24-week randomized, double-blind, placebo-controlled, Phase 3 study evaluating filgotinib on a background of conventional synthetic disease-modifying anti-rheumatic drug(s) (csDMARDs) among adult patients with moderately to severely active rheumatoid arthritis, who had not adequately responded to biologic DMARDs (bDMARDs). In this study, 23.7% of patients had received three or more bDMARDs. Patients were randomized (1:1:1) to receive filgotinib 100mg, filgotinib 200mg, or placebo. The primary endpoint was the proportion of patients achieving an ACR20 response at week 12. The safety profile was found to be consistent with previous trials. 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.



Non-responder imputation	Week 12 Placebo (n=148)[#]	Week 12 Filgotinib 100 mg (n=153)[#]	Week 12 Filgotinib 200 mg (n=147)[#]	Week 24 Placebo (n=148)[#]	Week 24 Filgotinib 100 mg (n=153)[#]	Week 24 Filgotinib 200 mg (n=147)[#]
ACR20 (%)	39.1	52.5**	66.0***	34.5	54.6***	69.4***
ACR50 (%)	14.9	32.8***	42.9***	18.9	38.3***	49.0***
ACR70 (%)	6.8	14.4*	21.8***	8.1	20.3***	32.0***
DAS28(CRP) < 3.2 (Low disease activity) (%)	8.8	22.2***	40.8***	10.8	27.9***	48.3***
DAS28(CRP) < 2.6 (Clinical remission) (%)	8.1	16.6**	32.8***	12.2	24.1**	33.6***

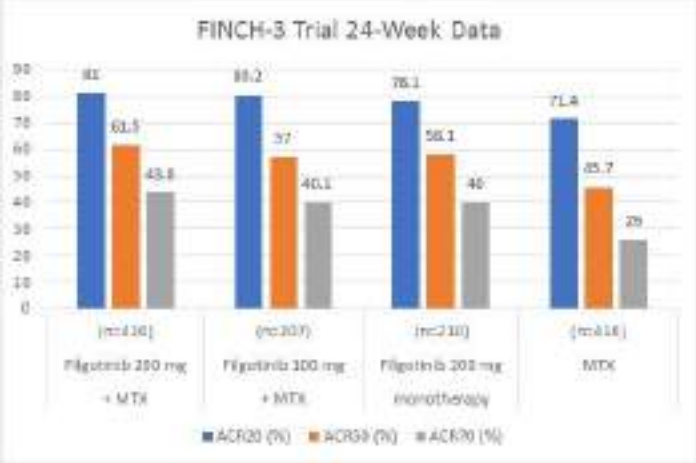
[#]Number of patients randomized to each treatment group and who received at least one dose of study drug

ACR20/50/70 represents American College of Rheumatology 20%/50%/70% improvements.

* p <0.05, compared to placebo
** p <0.01, compared to placebo
*** p <0.001, compared to placebo

Source: Gilead and Maxim Research

Exhibit 14. FINCH-3 trial data. FINCH 3 is 52-week randomized, double-blind, and active-controlled study examining filgotinib alone and in combination with MTX, enrolling N=1,252 adult patients with moderately to severely active RA who are naïve to MTX. Patients were randomized (2:1:1:2) to receive filgotinib 200mg plus MTX (n=417), filgotinib 100mg plus MTX (n=207), filgotinib 200mg alone (n=210), or MTX (n=418). The primary endpoint is the proportion of patients who achieve an ACR20 response at week 24. The safety profile of filgotinib in FINCH 3 is consistent with prior studies up to week 24. Serious adverse events occurred in 4.1%, 2.4%, 4.8%, and 2.9% of patients receiving filgotinib 200mg plus MTX, filgotinib 100mg plus MTX, filgotinib 200mg monotherapy, and MTX alone, respectively.



	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*	56.1[#]	45.7
ACR70 (%)	43.8***	40.1**	40.0[#]	29.0
DAS28(CRP) < 2.6 (Clinical remission) (%)	54.1***	42.5**	42.4[#]	29.1
HAQ-DI change	-0.94***	-0.90**	-0.88[#]	-0.79
mTSS change	0.28	0.22	0.64[#]	0.62

[§]Efficacy assessed at Week 24 for all endpoints

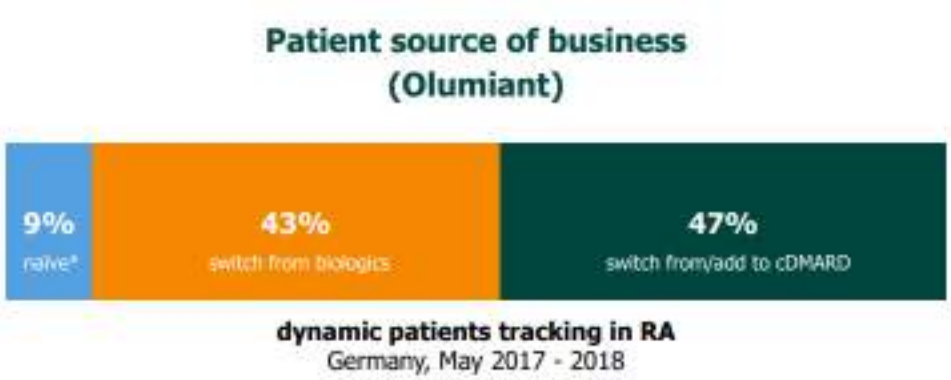
[#]Number of patients randomized to each treatment group and who received at least one dose of study drug

ACR20/50/70 represents American College of Rheumatology 20%/50%/70% improvements.

*** p <0.001, compared with MTX
* p < 0.05, compared with MTX
** p <0.01, compared with MTX
[#] Comparison not adjusted for multiplicity

Source: Gilead and Maxim Research

Exhibit 15. More than half of Olumiant RA patients are biologic naïve. When looking to the market potential of JAK inhibitors, a key data point is the patient demographics, in particular, how many are switching from bDMARDs and how many are biologic naïve. In the case of Olumiant in Germany, the data demonstrates that ~56% are either switching from a conventional DMARD such as methotrexate, or are treatment naïve. This is important because it demonstrates that physicians view JAK inhibitors as a treatment for the broader population, rather than just as an additional line of therapy in biologic-refractory patients. In Europe, in particular, JAK inhibitors have captured significant market share, accounting for as much as 20% of prescriptions. If similar success can be achieved in the US and Japan, there is a significant opportunity for second-generation JAK inhibitors like filgotinib and Rinvoq.



Source: Galapagos Corporate Presentation

Exhibit 16. FINCH program safety data. The short-term safety data from the FINCH program in RA has demonstrated a positive safety profile. In particular, the rate of serious infections was considerably lower than Humira treated patients, and only slightly higher than placebo, for the other safety endpoints, including thrombotic events, malignancy, deaths, and major adverse cardiac events, the filgotinib groups were on par with or less than placebo. One of the most important takeaways from the FINCH data (as well as the broader body of data on filgotinib) is that the 200mg dose appears to have safety similar to the 100mg dose, and similar to or lower than even placebo. This is important to note as the availability of two doses, the 100mg and 200mg, is key to the competitive proposition for filgotinib in RA.

N (%)	PBO/MTX	ADA 40 mg EOW	FIL 100 mg + MTX/cDMARDs	FIL 200 mg + MTX/cDMARDs	FIL 200 mg monotherapy	FIL total
	N=1039	N=325	N=840	N=1038	N=210	N=2088
serious infection	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 (0.2)*	0 (0)	1 (<0.1)
deaths	2 (0.2)	0 (0)	1 (0.1)	3 (0.3)	0 (0)	4 (0.2)
malignancy excl. 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)

Note: FINCH 1, 2, and 3 events up to week 24
*Excludes retinal vein occlusion observed in FINCH 2
FIL: filgotinib; ADA: adalimumab; MTX: methotrexate; PBO: placebo; csDMARD: conventional synthetic disease-modifying antirheumatic drug; DVT: deep vein thrombosis; PE: pulmonary embolism; NMSC: nonmelanoma skin carcinoma; MACE: major cardiovascular event
22

Galápagos

Source: Galapagos Corporate Presentation

Exhibit 17. DARWIN 3 longer-term safety data. The Phase 2b DARWIN program included a long-term extension (DARWIN 3) to measure the long-term safety data for filgotinib in RA. So far the company has released 156-week data, which includes 2,203 patient years of data. So far, the rates of adverse events have been notably low with serious infections only occurring at a rate of 1.2 per 100 PY, herpes zoster occurring at a rate of 1.5 events per 100 PY, and thrombotic events and deaths occurring at rates less than 1 per 100 PY.

event per 100 PYE	filgotinib 50-200 mg DARWIN 3 week 156
patient year exp.	2,203
serious infection	1.2
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*

Source: Galapagos Corporate Presentation

Modeling Assumptions in Rheumatoid Arthritis

1. We assume that Filgotinib launches for RA in 4Q20 in the EU and Japan, and in 2022 in the US.
2. We assume that there are ~1.5M RA patients in the US, ~2.3M in the EU, and ~1.25M in Japan.
3. We assume that 90% of RA patients will seek some form of DMARD during their lifetime based on the percentage of patients who have received methotrexate at some point.
4. We assume initial pricing will be on par with AbbVie's upadacitinib, which sells for $59k per year in the US and $13k in the EU. In japan we assume that pricing is 57% of the US price based on comparative average drug pricing.
5. We assume a 2% annual price increase for filgotinib.
6. We use a royalty-based revenue model for the US and Japan with 20%-30% tiered royalties from Gilead. In the EU, we model a 50% profit share for all territories.
7. We apply a 50% risk adjustment to the US based on the CRL and the need to refile and conduct the Type A meeting with the FDA.

Exhibit 18. Filgotinib – Rheumatoid arthritis market model (US).

Filgotinib - Rheumatoid Arthritis (US)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Patients in the US with RA	1,560,600	1,591,812	1,623,648	1,656,121	1,689,244	1,723,029	1,757,489	1,792,639
Increase in incidience	2%	2%	2%	2%	2%	2%	2%	2%
Patients Seeking Medication (90%)	1,404,540	1,432,631	1,461,283	1,490,509	1,520,319	1,550,726	1,581,740	1,613,375
Market Penetration				0.50%	0.75%	1.00%	1.25%	1.40%
Total Patients Treated				7,453	11,402	15,507	19,772	22,587
Cost of Treatment				60,000	61,200	62,424	63,672	64,946
Increase in Cost				2%	2%	2%	2%	2%
Gilead revenue ('000)				$ 447,153	$ 697,827	$ 968,025	$ 1,258,916	$ 1,466,950
Royalty				20%	22%	24%	26%	28%
Risk adjustment				50%	50%	50%	50%	50%
Total Revenue ('000)				**$ 44,715**	**$ 76,761**	**$ 116,163**	**$ 163,659**	**$ 205,373**

Source: Maxim Estimates

Exhibit 19. Filgotinib – Rheumatoid arthritis market model (EU).

Filgotinib - Rheumatoid Arthritis (EU)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
EU Population	515,338,170	516,884,185	518,434,837	519,990,142	521,550,112	523,114,763	524,684,107	526,258,159
Population Change	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
Patients in the EU with RA	2,369,460	2,416,849	2,465,186	2,514,490	2,564,780	2,616,075	2,668,397	2,721,765
Increase in incidience	2%	2%	2%	2%	2%	2%	2%	2%
Patients Seeking Medication (90%)	2,132,514	2,175,164	2,218,668	2,263,041	2,308,302	2,354,468	2,401,557	2,449,588
Market Penetration		0.04%	0.7%	1.25%	1.50%	1.60%	1.70%	1.80%
Total Patients Treated		816	15,531	28,288	34,625	37,671	40,826	44,093
Cost of Treatment		14,000	14,280	14,566	14,857	15,154	15,457	15,766
Increase in Cost		2%	2%	2%	2%	2%	2%	2%
Gilead revenue ('000)		$ 11,420	$ 221,778	$ 412,032	$ 514,414	$ 570,876	$ 631,060	$ 695,176
Profit Share		50%	50%	50%	50%	50%	50%	50%
Risk adjustment		0%	0%	0%	0%	0%	0%	0%
Total Revenue ('000)		**$ 5,710**	**$ 110,889**	**$ 206,016**	**$ 257,207**	**$ 285,438**	**$ 315,530**	**$ 347,588**

Source: Maxim Estimates

Exhibit 20. Filgotinib – Rheumatoid arthritis market model (Japan).

Filgotinib - Rheumatoid Arthritis (Japan)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Patients in the Japan with RA	1,252,431	1,258,693	1,264,987	1,271,312	1,277,668	1,284,056	1,290,477	1,296,929
Increase in incidience	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%
Patients Seeking Medication (90%)	1,127,188	1,132,824	1,138,488	1,144,180	1,149,901	1,155,651	1,161,429	1,167,236
Market Penetration		0.03%	0.4%	0.50%	0.75%	1.00%	1.25%	1.40%
Total Patients Treated		283	3,985	5,721	8,624	11,557	14,518	16,341
Cost of Treatment		33,630	34,303	34,989	35,688	36,402	37,130	37,873
Increase in Cost		2%	2%	2%	2%	2%	2%	2%
Gilead revenue ('000)		$ 9,524	$ 136,686	$ 200,167	$ 307,786	$ 420,682	$ 539,052	$ 618,892
Royalty		20%	20%	20%	22%	24%	26%	28%
Risk adjustment		0%	0%	0%	0%	0%	0%	0%
Total Revenue ('000)		**$ 1,905**	**$ 27,337**	**$ 40,033**	**$ 67,713**	**$ 100,964**	**$ 140,153**	**$ 173,290**

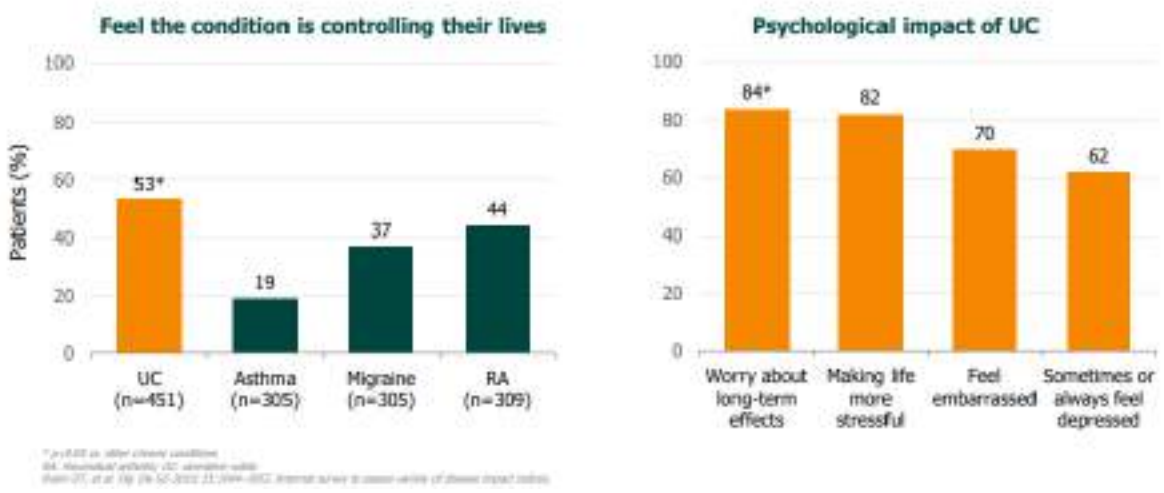
Source: Maxim Estimates

Inflammatory bowel disease. Inflammatory bowel disease (IBD) is an umbrella term for two specific conditions: Crohn's disease and ulcerative colitis. The reason these two conditions are grouped together is because they both involve chronic inflammation of the GI tract, and though the exact causes are unknown, immune dysfunction plays a large role in the pathogenesis and contributing factors can include diet, microbiota, breaches of the intestinal barrier, and genetics.[21,22,23] Both forms of IBD present symptoms including abdominal pain, diarrhea, rectal bleeding, severe internal crams, and weight loss. Anemia is also an associated complication, which makes a selective JAK inhibitor (specifically one that avoids JAK2-associated anemia) particularly attractive.[24] Overall, approximately 2M patients have IBD across the US and EU, of which 0.5M are treated with biologics such as TNF-inhibitors, creating a $9B market. IBD represents an attractive market for filgotinib as the options are more limited when compared to RA. IBD is expected to be a growing market over the next several years, nearing the size of the RA market by 2027.

Ulcerative colitis. Ulcerative colitis (UC) is a form of IBD that is specific to the colon, and can lead to inflammation and the formation of ulcers in the colon and rectum. The most common symptoms of the disease include diarrhea mixed with blood and mucus, often accompanied by abdominal pain. The chronic bleeding along with the inflammation leads to increased rates of anemia. Besides the impact on the bowels, patients with UC also often have extracolonic symptoms. These can include oral ulcers, inflammation of the iris, arthritis of the joints or spine, skin lesions, deep vein thrombosis, and a number of other symptoms. Like Crohn's disease, the causes of UC are not known, but a number of genetic and environmental factors are thought to play a role.

The disease can vary greatly in severity. Mild disease correlates with fewer than 4 bowel movements a day, no systemic signs of toxicity. Patients with mild disease may often believe that they are constipated when in fact they are experiencing tenesmus (the feeling of the need to empty the bowel leading to little-no fecal output). Patients with moderate disease experience more than 4 bowel movements per day but with minimal signs of toxicity. Patients may experience signs of anemia, though not requiring transfusion, as well as moderate abdominal pain and low-grade fever. Patients with severe disease have more than 6 bloody stools a day with observable massive and significant bloody bowel movement. Additionally, these patients present signs of toxicity such as fever, tachycardia, anemia, or elevated protein levels (erythrocyte sedimentation rate, ESR, or C-reactive protein, CRP). Fulminant UC is the most severe form of disease and correlated with 10+ bowel movements per day, continuous bleeding, toxicity, and colonic dilation. Blood transfusions are often required at this stage. Patients often have inflammation beyond the mucosal layer, impairing colonic motility and leading to toxic megacolon (a complication associated with severe acute dilation). Without treatment fulminant UC typically leads to death.

Exhibit 21. Disease burden in ulcerative colitis. Ulcerative colitis, and IBD more generally, is a particularly debilitating disease with 53% of patients feeling that the condition controls their lives, higher than asthma, migraine, and RA. The effects have a significant impact on quality of life (QoL) including increased stress, embarrassment, and even depression. The high impact on patient's quality of life highlights the unmet need in the market for additional treatments.



Source: Galapagos Corporate Presentation

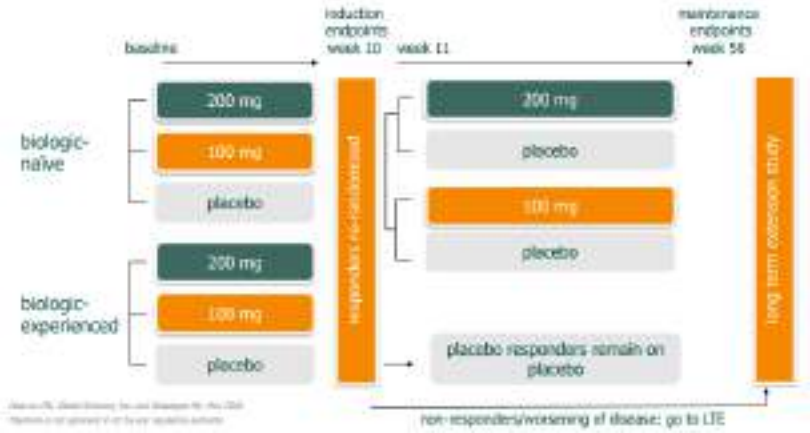
Treatment of UC. Unlike Crohn's disease, UC (at least the GI component) is curable through surgical removal of the large intestine or an ileo-anal pouch procedure. As these are both extremely invasive procedures, they are often reserved for severe cases of UC or events such as exsanguinating hemorrhage, frank perforation, carcinoma, or toxic megacolon. Fortunately there are a number of symptomatic treatments that

[21] Rashvand S, Behrooz M, Samsamikor M, Jacobson K, Hekmatdoost A (June 2018). "Dietary patterns and risk of ulcerative colitis: a case-control study". Journal of Human Nutrition and Dietetics. 31 (3): 408–412.
[22] Mukhopadhya I, Hansen R, El-Omar EM, Hold GL (February 2012). "IBD-what role do Proteobacteria play?". Nature Reviews. Gastroenterology & Hepatology. 9 (4): 219–30.
[23] Maloy KJ, Powrie F (June 2011). "Intestinal homeostasis and its breakdown in inflammatory bowel disease". Nature. 474 (7351): 298–306.
[24] Wang GF, Ren JA, Liu S, Chen J, Gu GS, Wang XB, Fan CG, Li JS (July 2012). "Clinical characteristics of non-perianal fistulating Crohn's disease in China: a single-center experience of 184 cases". Chinese Medical Journal. 125 (14): 2405–10.

largely focus on reducing inflammation. First line therapies include corticosteroids and aminosalicylates. Corticosteroids are used in a short-term setting, however, they are not safe for long-term use. Aminosalicylates have seen use in UC for decades. In particular, oral 5-aminosalicylic acid (5-ASA, mesalazine/mesalamine) drugs have been found to be particularly effective at inducing/maintaining remission in mild/moderate cases.[25] When 5-ASA drugs and corticosteroids fail to produce remission, TNF inhibitors such as infliximab, adalimumab, and golimumab have been found to be effective at producing remission, but only after other options have been exhausted due to side effects. The first oral therapy to be approved in UC was Pfizer's Xeljanz in 2018 establishing JAK inhibitors as a potential treatment in UC.[26]

Filgotinib in UC. Filgotinib has demonstrated positive data in UC from the P2b/3 SELECTION study (see data and trial design below, Exhibits 22-25). In the 10-week induction phase of the study, only the 200mg dose met the primary endpoint of clinical remission, while both doses achieved a remission in the 58-week maintenance phase. This result was poorly received by the market, which in our view was a misinterpretation of the data, likely based on conflating the effective dose in RA with effective dosing in UC. UC is a very different disease from RA, despite the underlying inflammation, so the lower dose being less effective compared to RA is not especially surprising. On the regulatory side, UC is handled by a different division in the FDA (Division of Gastroenterology and Inborn Errors vs. Division of Rheumatology and Transplant Medicine). There is precedent with JAK inhibitors specifically for a higher dose being approved for UC than the dose in RA. Pfizer's Xeljanz was approved for UC in 2018 with a 10mg BID dose during induction, followed by a 5mg or 10mg DIB dose, while its other previously approved indications (RA and PsA) are approved for a 5mg BID dose. This also reduces the risk related to the Type A meeting for filgo in RA, as a negative result for the 200mg dose in RA is not likely to read through to UC. Gilead/Galapagos is planning to file for approval in the EU and Japan around YE20, and in the US in mid-2021 following the result of the MANTA study, around the same time as refiling in RA.

Exhibit 22. Phase 2b/3 SELECTION trial. Gilead initiated the phase 2b/3 SELECTION study in UC in 4Q16. The P2b portion of the study completed in 2Q18 with N=350 patients, and following a planned futility analysis the data monitoring committee (DMC) recommended that the study continue into Phase 3 with both the 100mg and 200mg doses. The trial enrolled with N=1,351 patients and reported topline data in mid-2020. The design of the study is similar to the Phase 3 in Crohn's disease in that it used split data readouts for induction (10 weeks) and maintenance (58 weeks). Patients are randomized into either Filgotinib 100mg, filgotinib 200mg, or placebo and will be treated for 10 weeks. Patients who met response criteria at week 10 were moved into maintenance therapy and receive filgotinib or placebo for an additional 48 weeks. The primary induction endpoint was the proportion of patients achieving remission by the components of the Mayo Clinic Score (MCS) at week 10, while the maintenance endpoint is the same, but for week 58. A number of secondary endpoints are also measured including proportion of patients achieving an endoscopic sub score of zero, histologic remission, alternate MCS remission, and for the maintenance study, 6-month corticosteroid free remission.



Source: Galapagos Corporate Presentation

Exhibit 23. SELECTION induction study data. In the induction portion of the selection study, n=659 biologic-naïve patients and n=689 biologic experienced patients were enrolled. Among naïve patients receiving filgotinib 200mg, 26.1% of patients were considered responders compared to 15.3% for placebo. In experienced patients, 11.5% responded compared to 4.2% for placebo. Patients receiving filgotinib 100mg did not demonstrate a significant difference from placebo.

[25] Wang Y, Parker CE, Bhanji T, Feagan BG, MacDonald JK (April 2016). "Oral 5-aminosalicylic acid for induction of remission in ulcerative colitis". The Cochrane Database of Systematic Reviews. 4: CD000543.
[26] Commissioner, Office of the. "Press Announcements - FDA approves new treatment for moderately to severely active ulcerative colitis". www.fda.gov. Retrieved 31 May 2018.

Source: Galapagos Corporate Presentation

Exhibit 24. SELECTION maintenance study data. In the second phase of the study, the maintenance phase, included N=558 patients who demonstrated clinical remission or clinical response at 10 weeks in the induction study and were re-randomized 2:1 to their induction dose or placebo. By week 58, 37.2% of patients on filgotinib 200mg achieved clinical remission compared to 11.2% for placebo. For filgotinib 100mg, 23.8% of patients achieved clinical remission compared to 13.5% for placebo.

Source: Galapagos Corporate Presentation

Exhibit 25. SELECTION safety data. The safety profile for UC patients in the SELECTION study continues to demonstrate a positive safety profile, consistent with the prior body of data, with filgotinib groups experiencing rates of severe adverse events comparable to placebo. In the maintenance study, two deaths were observed in the 200mg group, though those were determined to be non-drug related, with one patient with pre-existing asthma experiencing asthma exacerbation and the other patient with pre-existing atherosclerosis experiencing left ventricular heart failure.

INDUCTION TRIAL SAFETY RESULTS

events	filgotinib 200 mg	filgotinib 100 mg	placebo
SAE in biologic-naïve patients	1.2%	4.7%	2.9%
SAE in biologic-experienced patients	7.3%	5.3%	6.3%

MAINTENANCE TRIAL SAFETY RESULTS

events	filgotinib 200 mg	placebo[1]	filgotinib 100 mg	placebo[2]
SAE	4.5%	-	4.5%	7.7%
deaths*	2	-	-	-

*Two deaths were observed in the filgotinib 200 mg treatment group in the maintenance trial, one patient with pre-existing asthma exacerbation, and the second patient with pre-existing atherosclerosis died due to left ventricular heart failure per autopsy report. Neither death was assessed as related to study drug by the investigator.

"Rates of serious infections, herpes zoster, venous thrombosis, pulmonary embolism and gastrointestinal perforation were low and comparable across treatment groups in both the induction and maintenance phases of the trial"

Source: Galapagos Corporate Presentation

Crohn's disease. Crohn's disease (CD) is a form of inflammatory bowel disease that affects any segment of the GI tract from the mouth to the anus. Though the cause is not known, CD seems to be due to a combination of genetic and environmental factors with a number of genetic risk factors contributing to the immune dysfunction.[27] The prevailing view on Crohn's is that it is driven by the adaptive immunity primarily a T-cell autoimmune disorder. Newer theories, however, have proposed involvement of impaired innate immunity driven by macrophages releasing

[27] Braat H, Peppelenbosch MP, Hommes DW (August 2006). "Immunology of Crohn's disease". Annals of the New York Academy of Sciences. 1072 (1): 135–54.

cytokines leading to a microbial-driven immune response in the colon where there is a high bacterial load.[28] In the US and Europe, CD has a prevalence of 3.2 per 1,000.[29]

Treatments for Crohn's disease. There is no cure for Crohn's disease, so the current goal of treatment is to achieve a clinical remission and prevent relapse. Despite the relatively high prevalence and market opportunity, under the current treatment paradigm, only around 10% of CD patients achieve a clinical remission. Injectable anti-TNF therapies have seen some success in CD, however a number of patients do not respond initially and as many as 50% of patients become refractory in a 1-year period.

Exhibit 26. Treatment options in Crohn's disease – Pricing and dosage forms.[30] Note that the pricing is based on 2018 numbers for a 70kg patient.

Drug (Brand)	Company (If Brand)	Mechanism	Dosing Form (Maintainence)	Annualized Cost Based on AWP Per Dose
Azathioprene		Inhibition of purine synthesis and DNA replication	Daily oral tablet	$ 6,200
Methotrexate		Inhibition of DNA synthesis and repair	Weekly intramuscular or subcutaneous injection	$ 8,400
Infliximab (Remicade)	Jassen Biotech	Anti-TNFα	8 week IV infusion	$ 31,900
Adalimumab (Humira)	AbbVie	Anti-TNFα	Biweekly subcutaneous injection	$ 76,000
Certolizumab (Cimzia)	UCB	Anti-TNFα	Monthly subcutaneous injection	$ 97,100
Vedolizumab (Ustekinumab)	Takeda	Inhibition of α4β7 integrin	8 week IV infusion	$ 38,100
Ustekinumab (Stelara)	Jassen Biotech	Inibition of IL-2 and IL-23	8 week subcutaneous injection	$ 133,800
Natalizumab (Tysabria)	Biogen	Inhibition of α4β1 and α4β7 integrin	4 week IV infusion	$ 80,300

Source: Maxim Research and Kish 2018

Ongoing DIVERSITY Phase 3 program. The DIVERSITY program is the Phase 3 program for filgotinib in the treatment of CD. Two trials are currently running; the DIVERSITY Trial, which was initiated in October 2016 by Gilead, and the DIVERSITYLTE long-term extension study, which was initiated in March 2017. DIVERSITY is investigating the safety and efficacy of 100mg and 200mg filgotinib once daily compared to placebo in moderately to severely active CD patients with a target enrollment of N=1,320. Patients also must have failed at least one prior antibody therapy. While women will be randomized to receive either 100mg or 200mg, men will only qualify for 200mg if they have failed at least one anti-TNF therapy and vedolizumab (anti-integrin antibody marketed by Takeda). The study is evaluating filgotinib for both induction and maintenance, so endpoints are split. The primary induction endpoints include proportion of patients achieving clinical remission by patient reported outcomes (PRO2) and proportion of patients achieving endoscopic response at week 10, and for maintenance, the endpoints are the same, except at week 58. Secondary endpoints include clinical remission rate by the Crohn's Disease Activity Index (CDAI) at weeks 10 and 58, patients achieving both clinical remission by PRO2 and endoscopic response at weeks 10 and 58, sustained clinical remission at both weeks 10 and 58, 6-month corticosteroid-free remission by PRO2 at week 58, as well as PK endpoints. Gilead is expected to complete enrollment in the trial by 1H21.

FITZROY Phase 2 data. The Phase 2 FITZROY trial enrolled N=174 patients with active CD to evaluate the 100mg or 200mg dose of filgotinib vs. placebo. N=130 patients received filgotinib compared to N=44 patients with placebo. The primary endpoint was clinical response as measured by CDAI score below 150 (from a starting point of 220-450) at 10 weeks. The trial hit the primary endpoint with 47% of patients achieving a clinical remission as compared to 23% for placebo (p=0.0077). Additionally, the number of patients achieving a 100-point reduction in CDAI scale at 10 weeks was significant at 60% for filgotinib vs. 41% for placebo. The trial was also stratified for patients with previous treatments and Galapagos reported that the response in TNF-naïve patients compared favorably compared to other treatments in other trials.

Other forms of Crohn's disease. Gilead is also developing filgotinib separately for a number of other sub-types of Crohn's disease, namely small bowel CD and fistulizing CD, both of which are in ongoing Phase 2 trials. Small bowel CD is Crohn's disease of the small intestine, which is associated with cramps, diarrhea, and weight loss, though occasionally patients may develop constipation instead of diarrhea. Fistulizing CD is a form of CD, which is associated with the formation of perianal fistulas. Fistulas are a common complication of Crohn's that affects as many as one-third of CD patients who will eventually develop at least one fistula. The cumulative frequency after 1 year is 12%,15% after 5 years, 21% after 10 years, and 26% after 20 years with the disease. The incidence also varies by type of CD. 92% of patients with colonic CD with rectal involvement present fistulas. Fistulas are an abnormal connection between two surfaces within the body, and the current hypothesis behind fistula formation in CD is that it is a result of epithelial-mesenchymal transition. The presence of fistulas is also correlated with serum levels of TNF-α, IL-6, and IL-12.[31]

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[29] Molodecky NA, et. al., (January 2012). "Increasing incidence and prevalence of the inflammatory bowel diseases with time, based on systematic review". Gastroenterology. 142 (1): 46–54.e42.
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[31] Scharl, Michael et al. "Fistulizing Crohn's Disease." Clinical and translational gastroenterology vol. 8,7 e106. 13 Jul. 2017, doi:10.1038/ctg.2017.33

Modeling Assumptions in Inflammatory Bowel Disease

1. We assume that filgotinib launches for UC in 2022 and CD in 2023 in the US, Japan, and EU.
2. We assume a prevalence of 238 per 100,000 for UC and a 201 per 100,000 for CD.
3. We assume initial pricing will be on par with AbbVie's upadacitinib which sells for $59K per year in the US and $13K in the EU. In japan, we assume pricing is 57% of the US price based on comparative average drug pricing.
4. We assume a 2% annual price increase for filgotinib.
5. We use a royalty-based revenue model for the US and Japan with 20%-30% tiered royalties from Gilead. In the EU, we model a 50% profit share for all territories.
6. We apply a 30% risk adjustment based on the stage of development.

Exhibit 27. Filgotinib – Inflammatory bowel disease market model (US).

Filgotinib - Inflammatory Bowel Disease (US)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
US Population	330,935,772	332,921,387	334,918,915	336,928,428	338,949,999	340,983,699	343,029,601	345,087,779
Population Change	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%
Patients in the US with Ulcerative Colitis (238 per 100,000)	787,627	792,353	797,107	801,890	806,701	811,541	816,410	821,309
Market Penetration				0.25%	0.75%	1.50%	1.75%	2.00%
Patients in the US with Crohns Disease (201 per 100,000)	665,181	669,172	673,187	677,226	681,289	685,377	689,489	693,626
Market Penetration					0.50%	1.00%	1.25%	1.50%
Total Patients Treated				2,005	9,457	19,027	22,906	26,831
Cost of Treatment				60,000	61,200	62,424	63,672	64,946
Increase in Cost				2%	2%	2%	2%	2%
Gilead revenue ('000)				$ 120,283	$ 578,750	$ 1,187,735	$ 1,458,469	$ 1,742,537
Royalty				20%	22%	24%	26%	28%
Risk adjustment				30%	30%	30%	30%	30%
Total Revenue ('000)				**$ 16,840**	**$ 89,128**	**$ 199,539**	**$ 265,441**	**$ 341,537**

Source: Maxim Estimates

Exhibit 28. Filgotinib – Inflammatory bowel disease market model (EU).

Filgotinib - Inflammatory Bowel Disease (EU)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
EU Population	516,585,622	518,135,378	519,689,784	521,248,854	522,812,600	524,381,038	525,954,181	527,532,044
Population Change	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
Patients in the EU with Ulcerative Colitis (238 per 100,000)	1,229,474	1,233,162	1,236,862	1,240,572	1,244,294	1,248,027	1,251,771	1,255,526
Market Penetration				0.25%	0.75%	1.50%	1.75%	2.00%
Patients in the EU with Crohns Disease (201 per 100,000)	1,038,337	1,041,452	1,044,576	1,047,710	1,050,853	1,054,006	1,057,168	1,060,339
Market Penetration					0.50%	1.00%	1.25%	1.50%
Total Patients Treated				3,101	14,586	29,260	35,121	41,016
Cost of Treatment				14,566	14,857	15,154	15,457	15,766
Increase in Cost				2%	2%	2%	2%	2%
Gilead revenue ('000)				$ 45,174	$ 216,710	$ 443,415	$ 542,864	$ 646,663
Profit Share				50%	50%	50%	50%	50%
Risk adjustment				30%	30%	30%	30%	30%
Total Revenue ('000)				**$ 15,811**	**$ 75,848**	**$ 155,195**	**$ 190,002**	**$ 226,332**

Source: Maxim Estimates

Exhibit 29. Filgotinib – Inflammatory bowel disease market model (Japan).

Filgotinib - Inflammatory Bowel Disease (Japan)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Population	125,994,506	125,742,517	125,491,032	125,240,050	124,989,570	124,739,591	124,490,111	124,241,131
Population Change	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%
Patients with Ulcerative Colitis (238 per 100,000)	299,867	299,267	298,669	298,071	297,475	296,880	296,286	295,694
Market Penetration				0.25%	0.75%	1.50%	1.75%	2.00%
Patients with Crohns Disease (201 per 100,000)	253,249	252,742	252,237	251,733	251,229	250,727	250,225	249,725
Market Penetration					0.50%	1.00%	1.25%	1.50%
Total Patients Treated				745	3,487	6,960	8,313	9,660
Cost of Treatment				34,989	35,688	36,402	37,130	37,873
Increase in Cost				2%	2%	2%	2%	2%
Gilead revenue ('000)				$ 26,073	$ 124,453	$ 253,376	$ 308,657	$ 365,842
Royalty				20%	22%	24%	26%	28%
Risk adjustment				30%	30%	30%	30%	30%
Total Revenue ('000)				**$ 3,650**	**$ 19,166**	**$ 42,567**	**$ 56,176**	**$ 71,705**

Source: Maxim Estimates

Psoriatic arthritis. Psoriatic arthritis (PsA) is a long-term inflammatory condition that occurs in ~30% of patients who have psoriasis and occurs in both children and adults. Though it typically is preceded by symptoms of psoriasis (red dry itchy and scaly skin), but in 15% of patients, the arthritis can actually precede skin symptoms.[32] The typical symptoms of PsA include pain and swelling in the joints, which are often red and inflamed. Asymmetrical oligoarthritis is present in 70% of cases vs. symmetrical arthritis, which is present in 15%. In addition to impacting the joints, PsA can affect the fingers/toes, nails, and skin. Dactylitis (sausage-like swelling of the fingers or toes) is also a common occurrence as are changes to the nails including pitting or separation from the nail bed. Though precise causes are not known, the disease is highly heritable, more so than RA, and environmental factors such as obesity, severe psoriasis, nail disease, and trauma are risk factors. CD8+ T cells are thought to play a role due to the association with HLA class I alleles, oligoclonal T-cell expansion, and the association of PsA in patients with HIV. Additionally, Type 17 cells have been found to be increased in the synovial fluid of PsA patients as compared with RA.[33] Recently, studies have also highlighted the role of cytokines such as IL-23 and IL17, as well as the TNF pathway in the pathogenesis of PsA.

Treatment options in PsA. Treatment in PsA is similar to other forms of inflammatory disease, with NSAID pain relievers such as naproxen indicated in mild forms of disease. For more severe cases, DMARDs are typically prescribed, however, trials have demonstrated only limited efficacy. Methotrexate, for example, was not found to be effective in a placebo-controlled trial.[34] Leflunomide has demonstrated efficacy for peripheral arthritis but not for psoriasis itself.[35] On the other hand, anti-TNF agents have demonstrated the ability to suppress both skin and joint inflammation as well as retarding radiographic progression. Interleukin blockers such as ustekinumab (targets p-40 subunit of IL-12 and IL-23), secukinumab, brodalumab, and ixekizumab (all three target IL-17), have also demonstrated efficacy in treating PsA with ustekinumab demonstrating more powerful treatment of skin symptoms vs. joints, and the IL-17 blockers improving both skin and musculoskeletal symptoms. JAK inhibitors have also been found to be effective in treating PsA. Xeljanz was approved for the indication in 2017 after demonstrating ACR20 in more than half of patients. AbbVie's Rinvoq was approved for PsA in June 2020 following the data from the SELECT-PsA Study for the 15mg dose after demonstrating ACR20 in 71% of patients.

Filgotinib in psoriatic arthritis - Phase 2 EQUATOR trial. A Phase 3 trial for filgotinib in PsA is currently being prepared. The drug was tested in the P2 EQUATOR Trial, which enrolled N=131 patients with moderately to severely active disease (defined as at least 5 swollen joints and at least 5 tender joints) and active or documented history of plaque psoriasis and insufficient response to previous treatment with conventional DMARDS. Patients were randomized 1:1 to receive either filgotinib 200mg or placebo for 16 weeks, and were evaluated using proportion of patients achieving ACR20 as the primary endpoint. A total of seven patients (five in the filgo group and two in placebo) discontinued the trial. At 16 weeks, 52 patients (80%) in the filgotinib group achieved ACR20 compared to 22 in the placebo group (33%). As for safety, 57% of patient in the filgotinib group experienced a treatment-related AE compared to 59% in the placebo group, supporting filgotinib's safety profile. One serious TEAE was reported in each group (pneumonia in filgotinib and hip fracture after fall in the placebo), though we note that the pneumonia was fatal.[36]

Rinvoq P3 data – SELECT-PsA 1 trial. The SELECT-PsA 1 study is Abbvie's Phase 3 study for upadacitinib (Rinvoq) in PsA. The study compared upadacitinib 15mg (n=429) and 30mg (n=423) to both active control (Humira) and placebo (n=423). The primary endpoint for the study was ACR20 at week 12. At week 12, 71% of patients receiving Rinvoq 15mg and 79% of patients receiving 30mg achieved ACR20, respectively, vs. 36% for placebo ($p<0.0001$). Compared to Humira, the 15mg dose achieved non-inferiority, while the 30mg dose achieved superiority. ACR50 was achieved by 38% and 52% of patients receiving 15mg and 30mg, respectively, vs. 13% of patients for placebo ($p<0.0001$). ACR70 was achieved by 16% and 25% of patients receiving 15mg and 30mg, respectively, vs. 2% of patients for placebo ($p<0.0001$). As for safety, serious infections occurred in 1.2% of 15mg patients and 2.6% of 30mg patients, vs 0.9% of placebo and 0.7% of Humira patients.

Ankylosing spondylitis. Ankylosing spondylitis (AS) is a form of arthritis that primarily affects the spine, though other joints can become involved (often the joints where the spine joins the pelvis). In advanced cases, this inflammation can lead to ankyloses, which is the formation of new bone in the spine, and can lead to spinal fusion. The hips and shoulder joints are also commonly involved and patients may experience eye and bowel problems. Although, like many inflammatory diseases, the precise cause is not known, it is thought to be a combination of environmental and genetic factors. In particular, a study of the UK ankylosing spondylitis population found that 90% of patients presented a specific human leukocyte antigen (HLA-B27). Individuals with the HLA-B27 genotype has a 1%-2% rate of developing AS, as compared to ~0.13% in the general US population (though prevalence studies have produced widely varying results).[37] AS is a systemic rheumatic disease and is related to IL-1 and TNFα. Additionally, CD8 and CD4 T cells are thought to play a role.

Treatment options in AS. Treatment of AS is typically directed at managing both pain and disease progression. On the pain side, NSAIDs and COX-2 inhibitors are often used, with the prescription NSAID indomethacin as the drug of choice. In terms of treating disease progression, DMARDs such as sulfasalazine and methotrexate have not been found particularly effective, and corticosteroids are not typically used due to a lack of evidence. Anti-TNF drugs have been found to produce substantial reductions in measures of disease activity. Humira for example (which

[32] Ritchlin, CT; Colbert, RA; Gladman, DD (March 2017). "Psoriatic Arthritis". New England Journal of Medicine (Review). 376 (10): 957–70.
[33] Leijten EF, et al. Brief report: enrichment of activated group 3 innate lymphoid cells in psoriatic arthritis synovial fluid. Arthritis Rheumatol 2015;67:2673-2678
[34] Kingsley GH, Kowalczyk A, Taylor H, et al. A randomized placebo-controlled trial of methotrexate in psoriatic arthritis. Rheumatology (Oxford) 2012;51:1368-1377
[35] Kaltwasser JP, Nash P, Gladman D, et al. Efficacy and safety of leflunomide in the treatment of psoriatic arthritis and psoriasis: a multinational, double-blind, randomized, placebo-controlled clinical trial. Arthritis Rheum 2004;50:1939-1950
[36] Philip Mease, MD , et al. "Efficacy and safety of filgotinib, a selective Janus kinase 1 inhibitor, in patients with active psoriatic arthritis (EQUATOR): results from a randomised, placebo-controlled, phase 2 trial" The Lancet, Vol 392, Issue 10162, p 2367-2377, December 01, 2018
[37] Khan, Muhammad Asim (2009). Ankylosing Spondylitis. Oxford University Press. p. 15.

is the #1 prescribed drug in AS) produced an improvement in symptoms of disease compared to 21% for patients taking placebo. In patients who don't respond to TNF-a inhibitors, the IL-17A inhibitor secukinumab is also an option.

Filgotinib in ankylosing spondylitis – Phase 2 TORTUGA trial. A Phase 3 trial for filgotinib in AS is currently being prepared. The Phase 2 TORTUGA study enrolled N=116 patients with ankylosing spondylitis, who did not adequately respond to two or more NSAIDS. Patients were randomized 1:1 to receive either filgotinib 200mg or placebo for 12 weeks, and were evaluated change from baseline in ankylosing spondylitis disease activity score (ASDAS) at week 12. Three patients from the filgotinib group and six from the placebo group did not complete the study. The change in ASDAS for the filgotinib patients was -1.47 compared to -0.57 for placebo, with a least squares mean difference of 0.85 between the two groups (P<0.0001). TEAEs were reported in 18 patients in each group and TEAE related discontinuation of the study occurred in two patients, on in the filgotinib group (grade 3 pneumonia) and one in the placebo group (high creatine kinase).[38]

Other diseases. In addition to the previously mentioned indications, filgotinib is also in earlier stage development (P2) for cutaneous lupus, lupus nephritis, Sjögren syndrome, and uveitis. The Phase 2 trials for cutaneous lupus and Sjögren's have read out, but both missed their primary endpoint in October 2019. Gilead commented that it did see a signal, but the path forward is currently unclear.

Lupus erythematosus is a collection of autoimmune diseases that cause the immune system to attack different body systems such as the joints, skin, and kidneys. Lupus is a relatively common condition with an estimated prevalence of ~5M worldwide. First-line therapy includes immunosuppressive drugs such as hydroxychloroquine and corticosteroids, with low-dose methotrexate as a second-line therapy.[39] In 2011, belimumab (a B-cell activating factor inhibitor) became the first new therapy approved in the US for systemic lupus erythematosus (SLE) in 50 years.[40] Cutaneous lupus is a specific for of lupus that may or may not be related to SLE, where the patient has symptoms restricted to the skin. Lupus has 4 different dermal manifestations: malar rash (butterfly rash on the cheeks), discoid rash (red patches on the skin), photosensitivity, and oral ulcers. Lupus nephritis is a form of inflammatory kidney disease that is cause by SLE. This disease can worsen over time and eventually lead to kidney failure. Lupus occurs at a rate of 2-7 per 10,000K.[41]

Sjögren syndrome (SjS) is a form of inflammatory disease affecting the body's moisture producing glands with symptoms often including dry eyes and dry mouth. Other symptoms include dry skin, vaginal dryness, chronic cough, muscle/joint pain, numbness in the extremities, and thyroid issues. The condition is thought to affect between 0.2% and 1.2% of the population and is more common in patients with other inflammatory conditions such as RA (30%-50%) and SLE (10%-25%).[42] Current treatments are typically symptomatic and supportive. For eye care, artificial tears can be used to ease dry eyes. Restasis can also be prescribed to treat the inflammation that disrupts tear production. Other prescription drugs such as Evoxac (cevimeline) are used to stimulate salivary flow. Systemic symptoms such as fatigue, joint pain, and neuropathy are often treated with biologic immunosuppressants such as rituximab and belimumab.

Uveitis is an inflammation of the uvea, the pigmented layer between the retina, and the outer fibrous layer of the eye composed of the sclera and cornea. The condition is estimated to affect 1 in 4,500 people between the ages of 20 and 60 and is often a cause for blindness; an estimated 10%-20% of blindness in the US is caused by uveitis.[43] Typical treatments include glucocorticoid steroid eye drops. Medications such as methotrexate can be used in recalcitrant or more aggressive cases. Though anti-TNF drugs are not approved for Uveitis, they see some use off-label.[44]

[38] Prof Désirée van der Heijde, MD, et al. "Efficacy and safety of filgotinib, a selective Janus kinase 1 inhibitor, in patients with active ankylosing spondylitis (TORTUGA): results from a randomised, placebo-controlled, phase 2 trial" *The Lancet,* Vol 392, Issue 10162, p 2378-2387, December 01, 2018.
[39] Böhm I (2004). "Increased peripheral blood B-cells expressing the CD5 molecules in association tp autoantibodies in patients with lupus erythematosus and evidence to selectively down-modulate them". Biomed Pharmacother. 58 (5): 338–43.
[40] "Systemic Lupus Erythematosus (SLE)" Centers for Disease Control and Prevention. https://www.cdc.gov/lupus/facts/detailed.html
[41] Danchenko, N.; Satia, J.A.; Anthony, M.S. (2006). "Epidemiology of systemic lupus erythematosus: a comparison of worldwide disease burden". Lupus. 15 (5): 308–318.
[42] John H., Klippel (2008). Primer on the rheumatic diseases (13th ed.). New York, NY: Springer. p. 389.
[43] Gritz DC, Wong IG (March 2004). "Incidence and prevalence of uveitis in Northern California; the Northern California Epidemiology of Uveitis Study". Ophthalmology. 111 (3): 491–500.
[44] Inês Leal, et. al. "Anti-TNF Drugs for Chronic Uveitis in Adults—A Systematic Review and Meta-Analysis of Randomized Controlled Trials." Front. Med., 24 May 2019

Modeling Assumptions in Other Indications

1. We assume that filgotinib launches for PsA and AS in 2023 in the US and EU, and for uveitis in 2025 in the US, EU, and Japan.
2. We assume a prevalence of 0.15% for PsA, 0.35% for AS, and 0.04% for uveitis.
3. We assume initial pricing of $59K per year in the US. As for EU, we assume that pricing will be approximately half the US price.
4. We assume initial pricing will be on par with AbbVie's upadacitinib, which sells for $59K per year in the US and $13K in the EU. In japan we assume pricing is 57% of the US price based on comparative average drug pricing.
5. We assume a 2% annual price increase for filgotinib.
6. We use a royalty-based revenue model for the US and Japan with 20%-30% tiered royalties from Gilead. In the EU, we model a 50% profit share for all territories.
7. We apply a 70% risk adjustment based on the stage of development.

Exhibit 30. Filgotinib – Other indications market model (US).

Filgotinib - Other Indications (US)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
US Population	330,935,772	332,921,387	334,918,915	336,928,428	338,949,999	340,983,699	343,029,601	345,087,779
Population Change	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%
Psoriatic Arthritis Prevalence (0.15%)					508,425	511,476	514,544	517,632
Ankylosing Spondylitis Prevalence (0.35%)					1,186,325	1,193,443	1,200,604	1,207,807
Uveitis Prevalence (0.04%)							130,351	131,133
Total Addressable Market	-	-	-	-	1,694,750	1,704,918	1,845,499	1,856,572
Market Penetration					0.20%	0.40%	0.60%	0.70%
Total Patients Treated					3,389	6,820	11,073	12,996
Cost of Treatment					61,200	62,424	63,672	64,946
Increase in Cost					2%	2%	2%	2%
Gilead revenue ('000)					$ 207,437	$ 425,711	$ 705,045	$ 844,038
Royalty					26%	28%	30%	30%
Risk adjustment					70%	70%	70%	70%
Total Revenue ('000)					**$ 16,180**	**$ 35,760**	**$ 63,454**	**$ 75,963**

Source: Maxim Estimates

Exhibit 31. Filgotinib – Other indications market model (EU).

Filgotinib - Other Indications (EU)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
EU Population	516,585,622	518,135,378	519,689,784	521,248,854	522,812,600	524,381,038	525,954,181	527,532,044
Population Change	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
Psoriatic Arthritis Prevalence (0.15%)					784,219	786,572	788,931	791,298
Ankylosing Spondylitis Prevalence (0.35%)					1,829,844	1,835,334	1,840,840	1,846,362
Uveitis Prevalence (0.04%)							199,863	200,462
Total Addressable Market					2,614,063	2,621,905	2,829,633	2,838,122
Market Penetration					0.20%	0.40%	0.60%	0.70%
Total Patients Treated					5,228	10,488	16,978	19,867
Cost of Treatment					14,857	15,154	15,457	15,766
Increase in Cost					2%	2%	2%	2%
Gilead revenue ('000)					$ 77,674	$ 158,930	$ 262,428	$ 313,226
Profit Share					50%	50%	50%	50%
Risk adjustment					70%	70%	70%	70%
Total Revenue ('000)					**$ 11,651**	**$ 23,839**	**$ 39,364**	**$ 46,984**

Source: Maxim Estimates

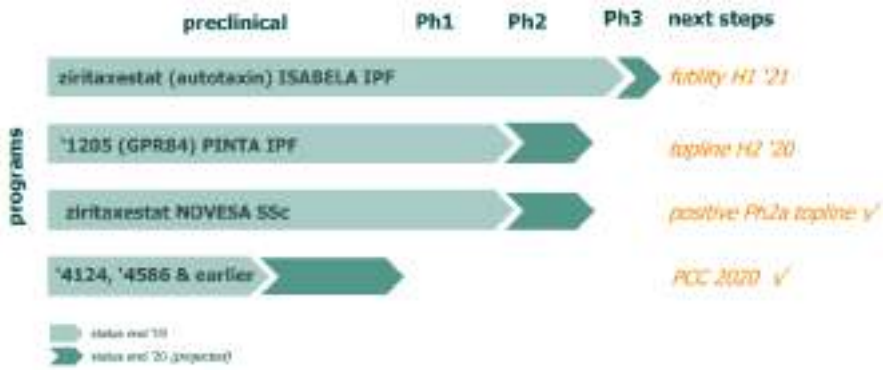
Exhibit 32. Filgotinib – Other indications market model (Japan).

Filgotinib - Other Indications (Japan)	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Population	125,994,506	125,742,517	125,491,032	125,240,050	124,989,570	124,739,591	124,490,111	124,241,131
Population Change	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%	-0.2%
Psoriatic Arthritis Prevalence (0.15%)					187,484	187,109	186,735	186,362
Ankylosing Spondylitis Prevalence (0.35%)					437,463	436,589	435,715	434,844
Uveitis Prevalence (0.04%)							47,306	47,212
Total Addressable Market					624,948	623,698	669,757	668,417
Market Penetration					0.20%	0.40%	0.60%	0.70%
Total Patients Treated					875	1,746	2,614	3,044
Cost of Treatment					35,688	36,402	37,130	37,873
Increase in Cost					2%	2%	2%	2%
Total revenue ('000)					$ 31,225	$ 63,571	$ 97,069	$ 115,281
Risk adjustment					70%	70%	70%	70%
Total Revenue ('000)					**$ 9,367**	**$ 19,071**	**$ 29,121**	**$ 34,584**

Source: Maxim Estimates

Idiopathic Pulmonary Fibrosis - GLPG1690 and GLPG1205

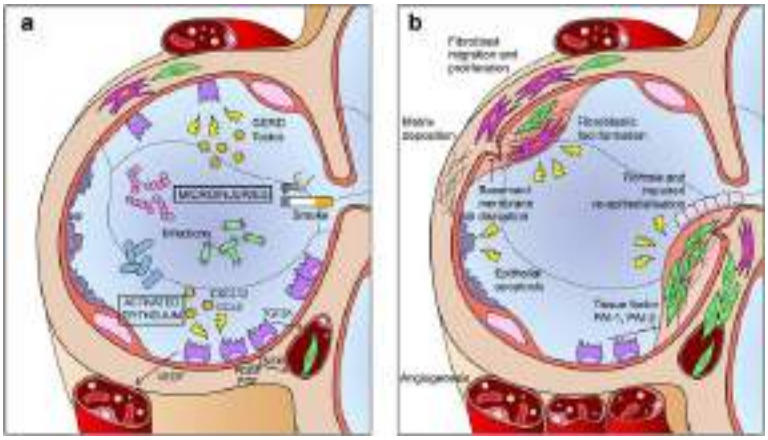
Exhibit 33. Building a fibrotic pipeline. Galapagos is developing a pipeline of anti-fibrotic drugs targeting treatment of idiopathic pulmonary fibrosis (IPF) and systemic sclerosis (SSc). The lead compound, GLPG1690, is an autotaxin inhibitor and entered Phase 3 development in 2H18 with the ISABELLA Trials (N=1500). GLPG1690 is also involved in an ongoing Phase 2 study called NOVESA in SSc which initiated in January 2019. The second compound in the fibrotic pipeline, GLPG1205, is a GPR84 inhibitor, which entered Phase 2 in October 2018 with the initiation of the PINTA Phase 2 trial. Beyond the two lead compounds, Galapagos has several early stage fibrosis programs (including collaborations with Fibrocor [private] in IPF and Evotec [FWB.EVT – NR] in an undisclosed fibrotic indication) and plans to explore additional fibrotic indications. The goal of this platform is to create a franchise in IPF with multiple potentially synergistic assets as well as combinations, similar to what Vertex did in cystic fibrosis. Considering the size of the IPF market and the level of unmet need, there is significant potential.



Source: Galapagos Corporate Presentation

Idiopathic pulmonary fibrosis. Idiopathic pulmonary fibrosis (IPF) is a fibrotic condition characterized by a progressive decline in lung function. The condition is irreversible and average life expectancy after diagnosis is 4 years and affects around 5 million people globally.[45] The actual cause of IPF is unknown, however, certain risk factors such as smoking, dust exposure (metal, wood, coal, biologic, etc.), and even viral infections have been found to increase the chances of developing disease.[46] IPF is believed to result from aberrant wound healing in the pulmonary interstitium with minimal associated inflammation. The current hypothesis states that the initial damage occurs in type 1 alveolar epithelial cells (AECs), which line the surface of the alveoli. When the type I AECs are injured, it is believed that type II AECs proliferate to replace them. In a healthy individual, the type II cells die and remaining cells differentiate into type I AECs, however, under pathologic conditions and in the presence of TGF-β, fibroblasts accumulate and differentiated into collagen secreting myofibroblasts.

Exhibit 34. IPF Pathogenesis. While the exact cause of IPF is not known, the condition is thought to be the result of an aberrant wound healing process occurring over repetitive injuries linked to smoking, environmental factors, or other medical conditions such as GERD, infections, or genetic predisposition. In the past, it was thought that inflammation was the first event which initiated scarring. However, more recent findings demonstrate that the fibroblastic foci precedes the inflammatory response and consequent collagen deposition (scarring/fibrosis).



Source: Sgalla et. al.

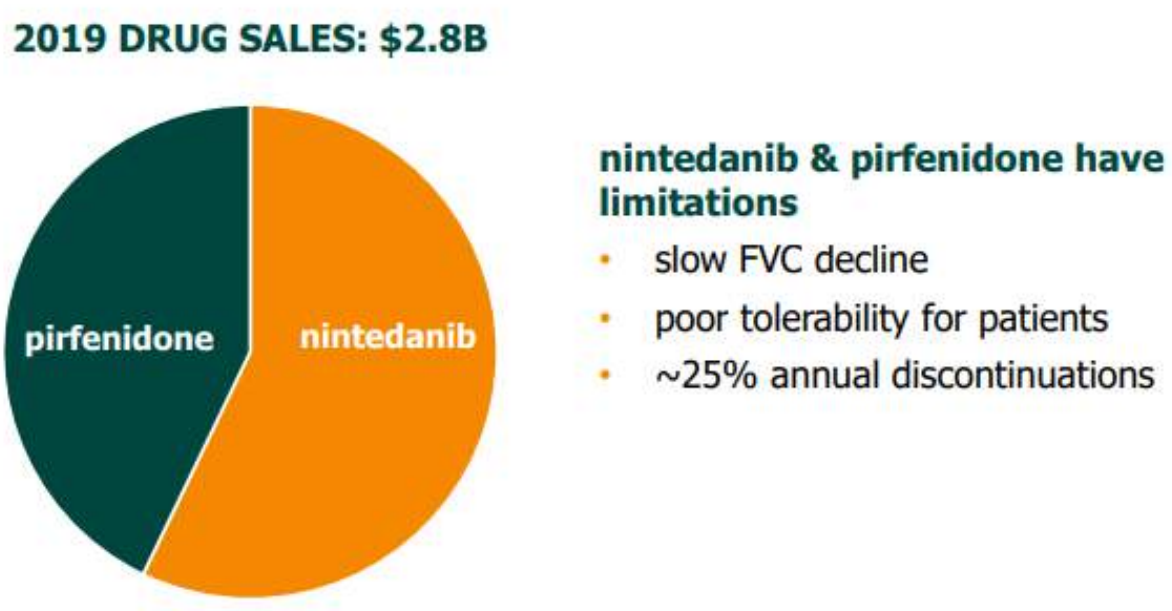
[45] "Idiopathic Pulmonary Fibrosis". NHLBI.
[46] Olson AL, Swigris JJ (Mar 2012). "Idiopathic pulmonary fibrosis: diagnosis and epidemiology". Clinics in Chest Medicine. 33 (1): 41–50.

Difficulty in drug development in IPF. Drug development in IPF has faced failures and difficulties. A number of drugs targeting the immune response and inflammation were evaluated since the first trial in IPF 30 years ago, however these were ultimately found to be ineffective. These treatment modalities, which included IFNγ-1β (cytokine based treatment for multiple sclerosis), bosentan (dual endothelin receptor antagonist used in the treatment of pulmonary artery hypertension), ambrisentan (endothelial receptor agonist used for pulmonary hypertension), and anticoagulants, were based on the inflammatory hypothesis and their failure called that idea into question. In October 2014, the FDA approved two antifibrotic drugs: nintedanib (Ofev) and pirfenidone (Esbriet). In 2017, a combination study by Boehringer Ingelheim (private) found a combination therapy consisting of both drugs to be safe, and demonstrated a signal of improved efficacy over Ofev alone, additional research is required to demonstrate efficacy.

Nintedanib. Nintedanib (Ofev) is an intracellular tyrosine kinase inhibitor (TKI) that targets vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and platelet-derived growth factor (PDGF) to decrease fibroblast proliferation, migration, and transformation.[47] In the Phase 3 INPULSIS trials, nintedanib was found to slow the annual reduction in forced vital capacity (FVC, total amount of air exhaled during the forced expiratory volume test). In INPULSIS-1 (N=513), the nintedanib 150mg BID group demonstrated a reduction of -114.7ml at week 52 compared to -239.9ml for placebo ($p<0.001$) and in INPULSIS-2 (N=548), nintedanib group demonstrated a reduction of -113.6ml at week 52 compared to -207.3ml for placebo ($p<0.001$). A key secondary endpoint was time to first acute exacerbation (sudden worsening of symptoms) as evaluated by site investigator. In the INPULSIS-1 study, there was no significant improvement in this endpoint, however, the INPULSIS-2 trial did see a hazard ratio improvement (0.38, P=0.005). The current dosing schedule for Ofev is 3x capsules a day.

Pirfenidone. Pirfenidone (Esbriet) is an established anti-fibrotic and inti-inflammatory drug that inhibits the production of transforming growth factor beta (TGF-β, a fibrotic mediator), reduces TGF-β stimulated collagen production, and reduces fibroblast proliferation. The drug was evaluated in three Phase 3 trials, the CAPACITY (study 004 and study 006) and the ASCEND Studies. In a pooled analysis of the three trials, a mean change from baseline in FVC of -216mL was found for pirfenidone compared to -363mL for placebo ($p<0.001$). We note, however, that in study 006, the difference between pirfenidone and placebo was non-significant. The current dosing schedule for Esbriet is 2x capsules a day.

Exhibit 35. Idiopathic pulmonary fibrosis market. There are two approved drugs in IPF: pirfenidone and nintedanib. Though these drugs make up a combined $2.8B market (up from $2.1B in 2018), there is room for improvement. Firstly, the drugs only slow the progression of the disease, though this is clinically important, it is less preferable compared to a drug which could stop disease progression (GLPG1690 reported stabilization after over the 12-week treatment period in the FLORA P2a study). Additionally, poor tolerability has led to a high discontinuation rate.



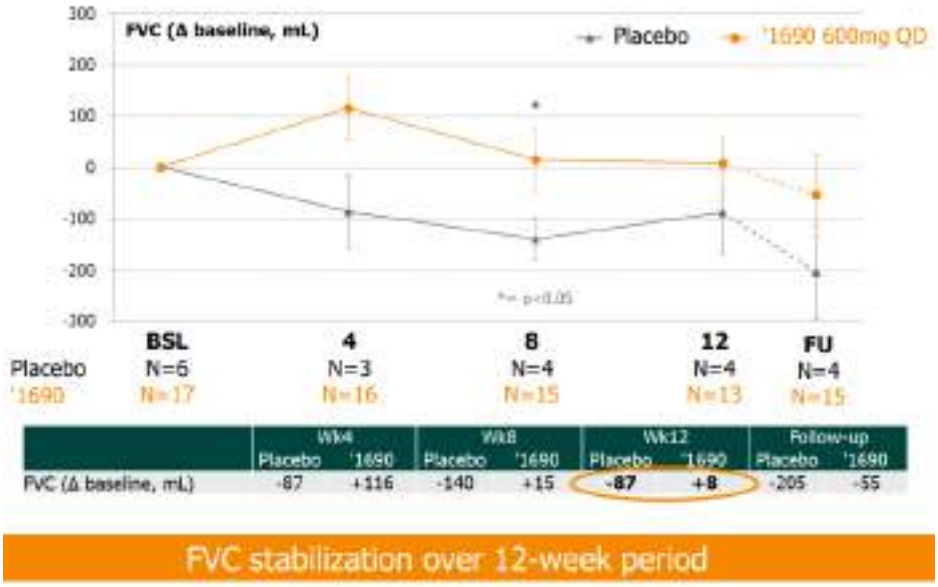
Source: Galapagos Corporate Presentation

[47] Brunton L, Knollman B, Hilal-Dandan R (2017-10-26). Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw Hill Professional.

Ziritaxestat (GLPG1690) – Autotaxin inhibition. Galapagos' most advanced clinical candidate in IPF is ziritaxestat, an orally active selective autotaxin inhibitor in Phase 3 development. Autotaxin (ATX) is an enzyme responsible for the production of lysophosphatidic acid (LPA), which has been found to be an important mediator of fibroblast recruitment and aberrant wound healing in IPF.[48] Increased ATX and LPA have been found to be associated with inflammatory and fibrotic conditions in the lung. Additionally, ziritaxestat has demonstrated an ability to reduce TGFβ-induced profibrotic cytokines such as IL-6, ET-1, and CTGF, which are central to the mechanisms of the current SoC drugs and actually demonstrated an additive effect in combination with nintedanib, which is promising for potential combination therapies.[49] The compound has demonstrated positive results in the FLORA P2a study in IPF, which reported a halt in disease progression, target engagement, and a favorable safety profile. The ongoing P3 ISABELLA program was initiated in December 2018 and is expected to support both NDA and MAA filings, an interim futility analysis is expected in 1H21 with topline data in early 2022. The drug is also in Phase 2 development for systemic sclerosis (SSc) with the NOVESA trial, which was initiated in January 2019 and demonstrated positive P2a data in 3Q20.

Phase 2a FLORA data. The Phase 2a FLORA trial was a 12-week randomized, double-blind, placebo controlled trial in IPF. N=23 patients were enrolled into the trial, 17 of whom received ziritaxestat 600mg once daily for 12 weeks, compared to 6 patients receiving placebo. The primary objective of the trial was safety, tolerability, and PK/PD, however, key secondary endpoints included evaluations of lung function (including change in FVC from baseline), changes in disease biomarkers to identify target engagement, functional respiratory imaging, and quality of life. The main inclusion criteria included biopsy-confirmed IPF and FVC≥50% predicted of normal. Over the 12-week period of the study, ziritaxestat patients demonstrated disease stability with no decline in FVC from baseline (actually a slight increase of +8mL) compared to -87mL for placebo. At the 2-week post-study follow-up, a decline of -55mL was found for the ziritaxestat group (after termination of treatment) compared to -205mL for placebo. Biomarker engagement was also observed in the study with plasma LPA 18:2 (the most common form of LPA) concentration reduction. As for safety and tolerability, TEAEs occurred in both groups similarly, with the most common events in the treatment group including lower respiratory tract infection, nasopharyngitis, cough, dyspepsia, and productive cough, which have previously been reported as frequent AEs in other IPF therapies.

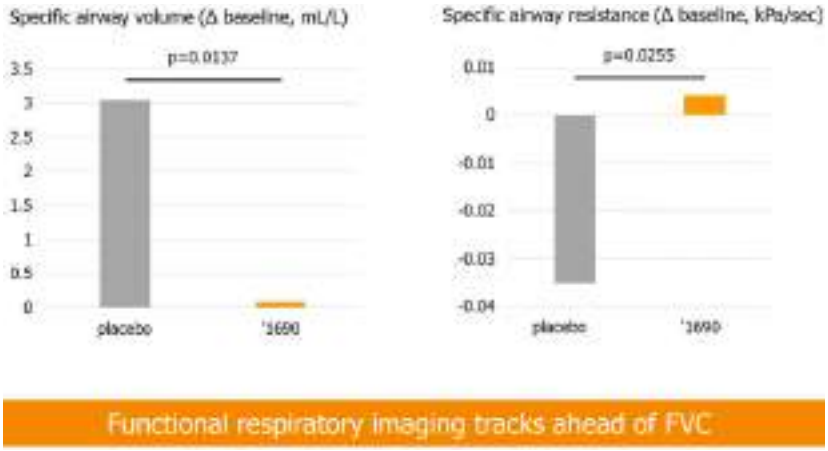
Exhibit 36. GLPG1690 FLORA P2a data. In the Phase 2a FLORA study, GLPG1690 demonstrated a stabilization of FVC decline over the 12-week treatment period. After the treatment period, there was a 2-week follow-up. Patients treated with GLPG1690 during the main portion of the study experienced a -55mL decline from baseline, compared to -205mL for placebo.



Source: Galapagos Corporate Presentation

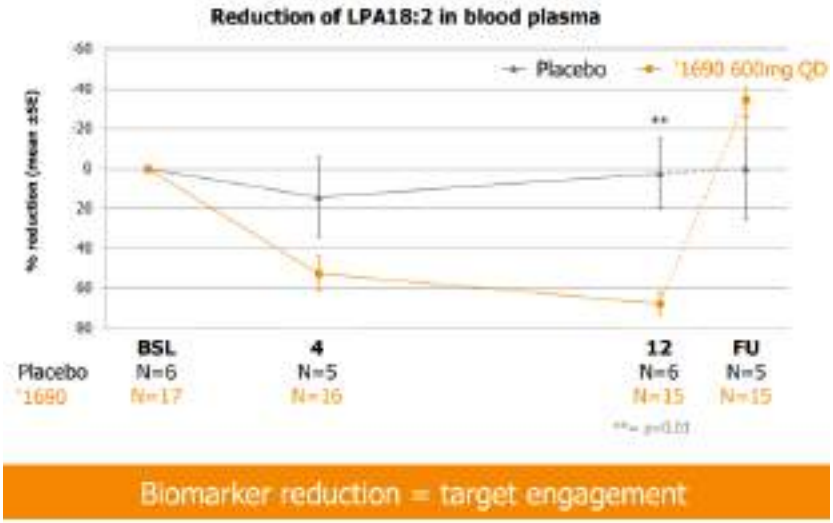
[48] Tager A, LaCamera P, Shea B, Campanella G, Selman M, Zhao Z, et al. The lysophosphatidic acid receptor LPA1 links pulmonary fibrosis to lung injury by mediating fibroblast recruitment and vascular leak. Nat Med. 2008;14:45–54.
[49] Autotaxin Inhibitor GLPG1690 Affects TGFβ-induced Production of the Pro-Fibrotic Mediators CTGF, IL-6 and ET-1 in Fibroblasts, ATS 2017

Exhibit 37. Functional respiratory imaging indicates disease stabilization. A key secondary endpoint was the functional respiratory imaging results. Increased airway volume and specific airway resistance are markers of disease progression. In both cases, the change from baseline for the GLPG1690 group was significantly lower than that of the placebo group.



Source: Galapagos Corporate Presentation

Exhibit 38. Biomarker data indicates target engagement. Biomarker data in the FLORA trial was an important endpoint to confirm the mechanism of action for GLPG1690. The data demonstrate a sustained reduction of the plasma concentration of LPA 18:2 and that once daily dosing is sufficient to maintain reduced levels. Additionally, the effect was found to be reversible, returning to normal levels at the 2-week follow-up.



Source: Galapagos Corporate Presentation

Exhibit 39. GLPG1690 Phase 3 program in IPF– ISABELA 1 & 2. The ongoing global Phase 3 ISABELLA program (N=1500) consists of two identical trials, the ISABELA 1 and ISABELLA 2 trials. Patients will be randomized to receive GLPG1690 on top of their SoC therapy whether or not they were previously or currently are treated with Esbriet (pirfenidone) and Ofev (nintedanib). The primary endpoint will be the rate of decline in FVC in mL until week 52. Patients will continue treatment until the last patient in the study completes 52-week follow-up, this will allow for longer term evaluation for patients enrolled early in the study and for evaluation of less frequent disease events. Secondary endpoints will include respiratory-related hospitalizations, mortality, quality of life, safety, and tolerability. An interim futility analysis is planned for 1H21, with topline data expected in 2022.

Source: Galapagos Corporate Presentation

GLPG1205 – GPR84 inhibition. Galapagos has a second candidate in its antifibrotic pipeline, GLPG1205, a GPR84 inhibitor. GPR84 (probable G-protein coupled receptor 84) is a member of the metabolic G-protein coupled receptor family that is found primarily on immune cells (bone marrow cells, splenic T and B cells, and circulating granulocytes/monocytes/macrophages). Activation of GPR84 has been found to play a role in the inflammatory response and was thus initially targeted for development as an anti-inflammatory. However, more recently, it has been demonstrated to play a role in fibrotic pathways involving macrophages, fibroblasts, and epithelial cells. Inhibition of GPR84 in mouse models has demonstrated reductions in kidney, lung, heart, liver, pancreas, and skin fibrosis. Additionally, GPR84 knockout models of mice demonstrated reduced kidney fibrosis in models of adenine-induce nephropathy.[50] GLPG1205 has been tested in preclinical studies, demonstrating a positive reduction in signs and symptoms of IPF has demonstrated a positive safety/tolerability profile in previous studies in ulcerative colitis.

Exhibit 40. GLPG1205 Phase 2 program in IPF – PINTA. The Phase 2 PINTA trial is a randomized double-blind, placebo-controlled trial investigating the 100mg dose of GLPG1205. Dosing will continue for 26 weeks in up to N=60 patients with diagnosed IPF. Similarly to the ISABELLA trials, patients may also continue their SoC background therapy. The primary objective of the study is the change in FVC over the 26 weeks. Secondary endpoints will include respiratory imaging, time to major disease events, changes in functional exercise capacity, QoL, PK/PD, and safety and tolerability. The trial will include patients from across Europe, North Africa, and the Middle East. Dosing initiated in October 2018 and the trial is fully recruited with topline data expected in 2H20.

[50] Lyne Gagnon, et al. "A Newly Discovered Antifibrotic Pathway Regulated by Two Fatty Acid Receptors" *American Journal of Pathology*, May 2018 Volume 188, Issue 5, Pages 1132–1148. https://ajp.amjpathol.org/article/S0002-9440(17)30804-0/fulltext

Source: Galapagos Corporate Presentation

Systemic sclerosis (SSc) Phase 2 program. In addition to the IPF program, Galapagos is developing GLPG1690 in systemic sclerosis, a severe autoimmune rheumatic disease that affects approximately 95K-155K patients across the US and EU. The characteristic symptom of SSc is hardening and scarring of the skin, which can appear tight, reddish, and scaly. If large areas are affected, fat and muscle wastage can weaken limbs. There are two major subgroups of SSc depending on the extent of skin symptoms. Limited SSc affects the skin below the elbows and knees with or without face symptoms while diffuse SSc also affects the skin above the elbows and knees and can affect the torso. In the 35% of patients with diffuse disease, visceral organs can be impacted by the fibrotic processes as well including the lungs, kidneys, heart, and GI tract. SSc is among the more lethal rheumatic diseases with a 10-year survival rate of 55% in diffuse disease, while patients with limited disease have a 75% 10-year survival rate.

Treatments for SSc. Therapeutic options of SSc remain limited, mostly focused on symptomatic relief including reducing inflammation and softening the skin. Topical treatments for the skin changes are available which do not alter the disease course. Painful symptoms can be treated with NSAIDs and skin tightness can be treated with methotrexate or cyclosporine.[51] As for organ-related symptoms, the occurrence of acute renal failure and hypertension can be effectively treated with ACE inhibitors, even in those patients who require dialysis.[52] Nintedanib was actually approved in September 2019 for the treatment of pulmonary decline in patients with SSc associated with interstitial lung disease.

Exhibit 41. GLPG1690 Phase 2a program in diffuse cutaneous SSc – NOVESA. The double-blind placebo-controlled NOVESA trial was initiated in early 2019 to evaluate the safety and efficacy of GLPG1690 on SSc patients. The trial enrolled N=33 patients with diffuse disease and multiple organ involvement and is recruiting patients from the US and EU. The primary endpoint of the trial was the modified Rodnan Skin Score (mRSS) at 24 weeks. Secondary outcome measures included FVC, QoL assessments, safety and tolerability.

> **Topline data from the NOVESA study.** The primary endpoint for the NOVESA was change in mRSS from baseline at week 24. N=33 patients were evaluated in the study, most of whom had a background of immunosuppressant therapy. For the patients receiving ziritaxestat 600mg (n=21), a change of -8.3 was observed, compared to -5.7 for placebo (n=12, p=0.0411). Overall, the drug was well tolerated with no deaths in the trial. SAEs occurred in 2 patients in the treatment group compared to one in the placebo group. Both patients in the treatment group recovered and continued onto the open label extension. After completion, 94% of patients who completed the NOVESA trial continued into the long term open label extension trial.

Source: Galapagos Corporate Presentation

[51] Zandman-Goddard G, Tweezer-Zaks N, Shoenfeld Y (2005). "New therapeutic strategies for systemic sclerosis--a critical analysis of the literature". Clin. Dev. Immunol. 12 (3): 165–73.
[52] Ibid.

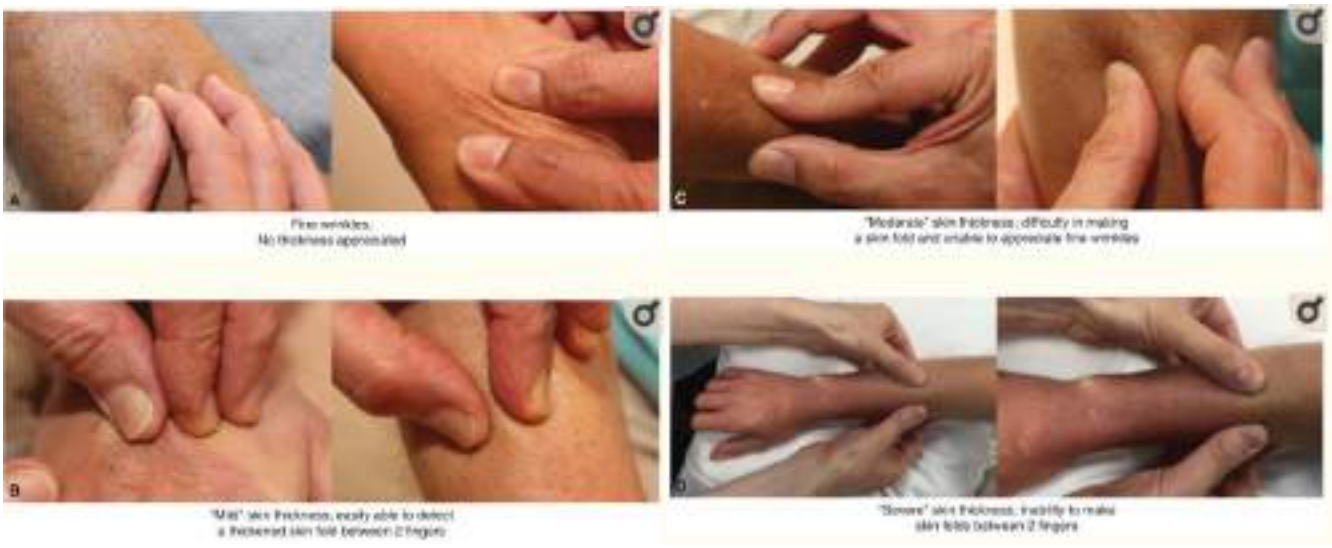
Exhibit 42. Modified Rodnan skin score. The modified Rodnan skin score (mRSS) is a measure of skin thickness commonly used in SSc trials that uses the ability to assess wrinkles via skin palpitation as a measure of thickness. The score is a sum of the presence of thick skin on 10 cutaneous areas (fingers, hands, forearms, upper arms, face, anterior chest, abdomen, thighs, legs, and feet) independently assessed for the left and right hand sides (except for face, anterior chest, and abdomen) which are graded on a scale of 0 (no thickening) to 3 (severe thickening).

A) mRSS = 0 – No thickening: Fine wrinkling occurs when the skin is pinched indicating no thickening.

B) mRSS = 1 – Mild thickening: Easy to detect a thickened skin fold between two fingers.

C) mRSS = 2 – Moderate thickening: A skin fold is difficult to make between two fingers, inability to appreciate fine wrinkles.

D) mRSS = 3 – Severe thickening: Inability to make a skin fold between two fingers.



Source: Khanna et al. (2017)

Modeling Assumptions in Idiopathic Pulmonary Fibrosis

1. We assume that GLPG1690 launches for IPF in 2023 in the US and EU.
2. We assume a prevalence of 30 per 100K in the US and 23 per 100K in the EU.
3. We assume initial pricing of $100K per year in the US on par with Ofev initial pricing and $50K in the EU, on par with Esbriet initial pricing in EU.
4. We assume a 2% annual price increase.
5. We use a royalty-based revenue model for the US with low 20% tiered royalties from Gilead in the US. In the EU, we model Galapagos commercializing the product internally.
6. We apply a 30% risk adjustment based on the stage of development.

Exhibit 43. GLPG1690 – Idiopathic pulmonary fibrosis market model (US).

GLPG1690 in Ideopathic Pulmonary Fibrosis (US)	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
US Population	328,962,000	330,935,772	332,921,387	334,918,915	336,928,428	338,949,999	340,983,699	343,029,601	345,087,779
Population Change	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%	0.6%
Patients in the US with IPF (30 per 100,000)	98,689	99,281	99,876	100,476	101,079	101,685	102,295	102,909	103,526
Market Penetration						4.00%	8.00%	15.00%	22.00%
Total Patients Treated						4,067	8,184	15,436	22,776
Cost of Treatment						100,000	102,000	104,040	106,121
Increase in Cost						2%	2%	2%	2%
Gilead revenue ('000)						$ 406,740	$ 834,728	$ 1,605,996	$ 2,416,985
Royalty						20%	21%	21%	22%
Risk adjustment						30%	30%	30%	30%
Total Revenue ('000)						**$ 56,944**	**$ 122,705**	**$ 236,081**	**$ 372,216**

Source: Maxim Estimates

Exhibit 44. GLPG1690 – Idiopathic pulmonary fibrosis market model (EU).

GLPG1690 in Ideopathic Pulmonary Fibrosis (EU)	2018E	2019E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
EU Population	515,542,000	517,088,626	518,639,892	520,195,812	521,756,399	523,321,668	524,891,633	526,466,308	528,045,707
Population Change	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%	0.3%
Patients in the EU with IPF (23 per 100,000)	118,575	118,930	119,287	119,645	120,004	120,364	120,725	121,087	121,451
Market Penetration						2.00%	8.00%	12.00%	17.00%
Total Patients Treated						2,407	9,658	14,530	20,647
Cost of Treatment						50,000	51,000	52,020	53,060
Increase in Cost						2%	2%	2%	2%
Total revenue ('000)						$ 120,364	$ 492,558	$ 755,875	$ 1,095,516
Risk adjustment						30%	30%	30%	30%
Total Revenue ('000)						**$ 84,255**	**$ 344,791**	**$ 529,113**	**$ 766,861**

Source: Maxim Estimates

Toledo Platform

Toledo program. Toledo is a codename for a novel target class discovered by Galapagos for development in multiple inflammatory diseases. The platform targets salt-inducible kinase (SIK). SIKs phosphorylate HDAC and CRTCs, which block NFkB and promote CREB, respectively. The result of blocking this activity is promotion of anti-inflammatory (healing cytokines) and reducing inflammatory cytokines. Galapagos has developed several compounds inhibiting this this target family, which have demonstrated unprecedented activity in certain preclinical inflammatory models. By rebalancing the immune system, rather than just suppressing it as traditional anti-inflammatory drugs do, Toledo is likely to avoid or reduce many of the side effects associated with treatments for inflammatory diseases. It also may allow a higher dose than traditional anti-inflammatory disease as it works on both sides of the immune balance.

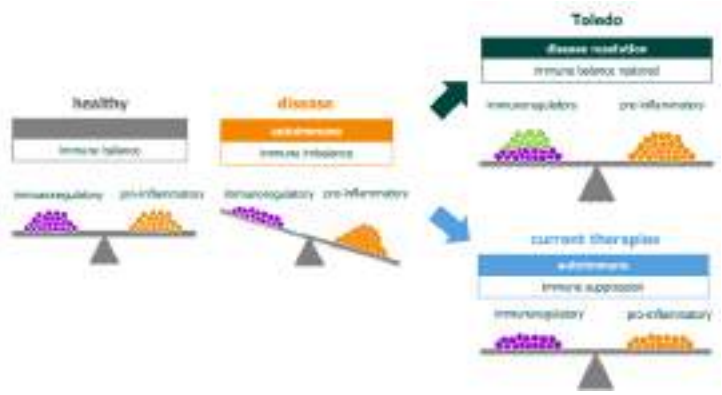
The strategy for the Toledo program is to advance multiple candidates with different selectivity profiles, and then to take these through a number of *in vivo* models for different inflammatory diseases including IBD, RA, psoriasis, systemic lupus erythematosus, OA, osteoporosis, and fibrosis. The first two generations of the Toledo compound, GLPG3312 (pan-TOL) and GLPG3970 (TOL2/3), have entered clinical development and a third candidate has been selected, GLPG4399 (TOL3). GLPG3970 was selected to move forward and has recently entered a P1 study in psoriasis, a P2 study in ulcerative colitis, and a P2 study in rheumatoid arthritis. Two additional P2 studies are planned for initiation in 1H21. Data from these proof-of-concept studies are expected to be reported in mid-2021.

Exhibit 45. Toledo pipeline. Toledo compounds are being developed across a number of inflammatory conditions including inflammatory bowel disease (IBD), rheumatoid arthritis (RA), psoriasis (Pso), psoriatic arthritis (PsA), systemic lupus erythematosus (SLE), osteoarthritis (OA), osteoporosis (OP), and fibrosis (FIB). The first two compounds (GLPG3312 and GLPG3970) have entered clinical development, and a third and fourth have been selected, and a fifth is in lead optimization.

GLPG3970 – The second-generation Toledo compound has demonstrated preclinical results across multiple inflammatory/fibrotic disorders. GLPG3970 has demonstrated its dual mode of action in a Phase 1 trial. The trial, named CALOSOMA, is a 2-part study in psoriasis. The first part was a single and multiple ascending dose study in N=52 healthy adult males which evaluated the PK/PD and safety in N=52 healthy adults. The second part, which recently initiated enrollment, involves N=25 moderate to severe psoriasis patients, who will be dosed for 6 weeks. Two phase 2 trials are also ongoing. The SEA TURTLE trial in ulcerative colitis is evaluating efficacy, safety, tolerability, and PK/PD in N=30 subjects with moderate-severe UC. The trial is double blind placebo controlled and has a primary endpoint of change from baseline in Mayo clinic score. The LADYBUG trial in RA is also a double blind placebo controlled trial, which will enroll N=25 patients with insufficient response to methotrexate. Patients will be dosed daily for 6 weeks with a primary endpoint of change in baseline of DAS28 CRP at week 6.

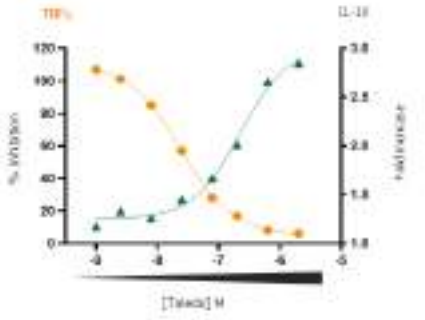
Source: Toledo Roundtable Presentation

Exhibit 46. Toledo platform restores the immune balance. In treatment of inflammatory disease, the goal is to restore an immune imbalance. Most current therapies focus on reducing the presence of pro-inflammatory markers (think TNFα). Toledo is differentiated in that it focuses on increasing the presence of immunoregulatory signals. This approach is similar to what is thought to make cell therapy effective in treatment of inflammatory conditions.



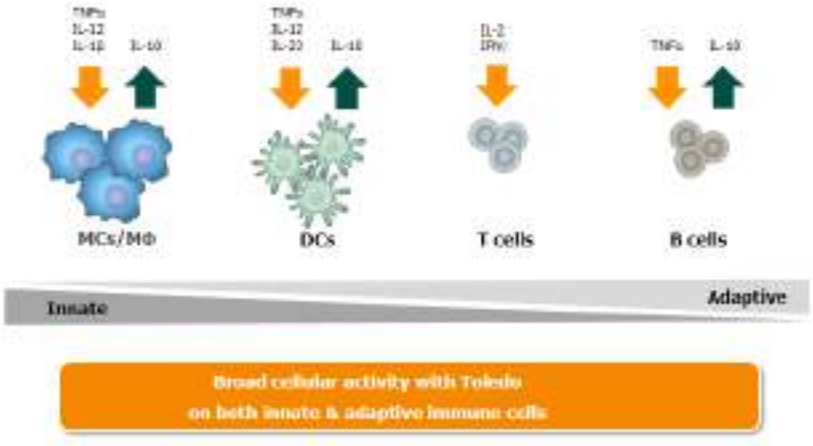
Source: Galapagos Corporate Presentation

Exhibit 47. Toledo promotes anti-inflammatory cytokines and reduces pro-inflammatory cytokines. As concentration of Toledo increases, there is a reduction to pro-inflammatory cytokines such as TNFα (the target for Humira), which increases the concentration of anti-inflammatory cytokines (healing cytokines) such as IL-10



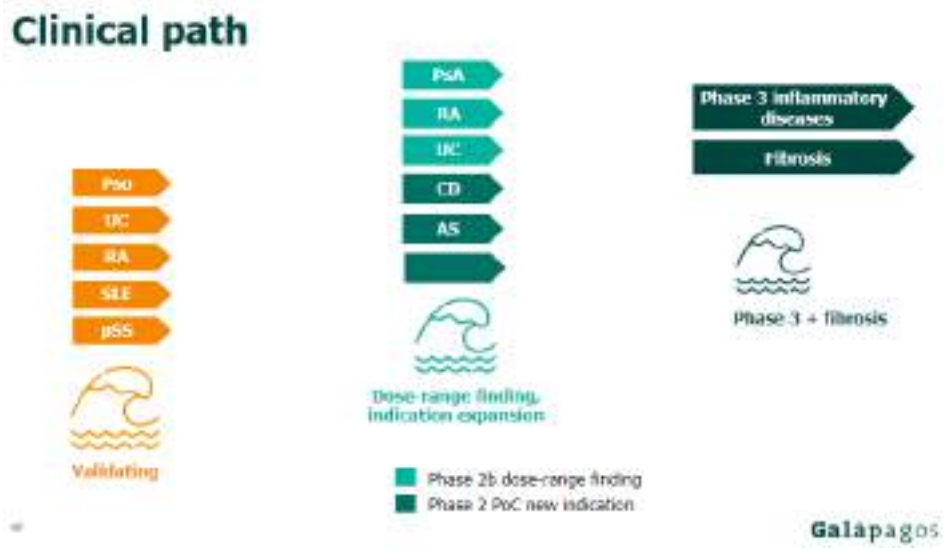
Source: Toledo Roundtable Presentation

Exhibit 48. Activity of Toledo acts across the immune system. One of the key benefits to Toledo is that it has broad-reaching activity on multiple cytokines. This means that it can act across the immune system to impact cells in the innate immune system such as macrophages (suppressing the M1 inflammatory phenotype and promoting the M2 anti-inflammatory phenotype) and dendritic cells, as well as the adaptive immune system with T cells and B cells.



Source: Toledo Roundtable Presentation

Exhibit 49. Clinical pathway for Toledo. The pathway for Toledo is based on initiating multiple small-scale trial validation studies across multiple indications and then moving into larger dose-ranging studies in P2b, as well as adding proof-of-concept studies for new indications. The next step is moving into P3 for inflammatory diseases and evaluating the compound(s) in fibrotic conditions.



Source: Toledo Roundtable Presentation

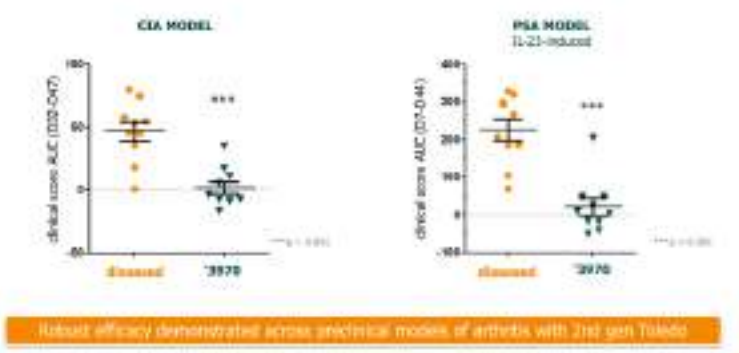
Exhibit 50. Multiple proof-of-concept studies planned and ongoing. The lead Toledo asset is GLPG3970. Currently, three proof-of-concept studies are ongoing in psoriasis, UC, and RA. The next step includes more challenging indications such as SLE and Sjörgren's syndrome.

Source: Toledo Roundtable Presentation

Exhibit 51. GLPG3970 proof-of-concept studies. The three ongoing proof-of-concept studies are the P1b CALSOMA study in psoriasis, P2 SEA TURTLE study in UC, and the P2 LADYBUG study in RA. The CALSOMA study involves N=25 patients with moderate to severe psoriasis and is evaluating the safety and efficacy of GLPG1690 over 6 weeks of treatment with a 2-week follow-up. The SEA TURTLE study in UC is evaluating N=30 treatment experienced patients with moderate to severe UC for 6 weeks with a 2-week follow-up. The LADYBUG study is evaluating N=25 patients with moderate to severe RA who have had inadequate response to MTX for 6 weeks with a 2-week follow-up.

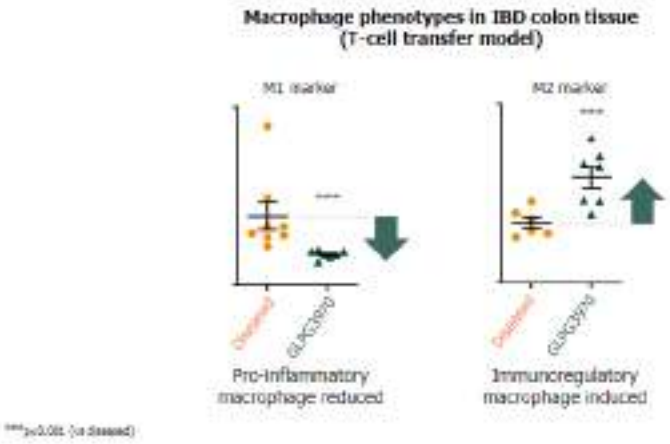
Source: Toledo Roundtable Presentation

Exhibit 52. GLPG3970 Preclinical Data in arthritis. GLPG3970 has demonstrated statistically significant improvements (p<0.001) in models of both collagen-induced arthritis and psoriatic arthritis.



Source: Galapagos Corporate Presentation

Exhibit 53. GLPG3970 preclinical *in vivo* data demonstrates both sides of the immune balance. In *in vivo* studies, GLPG3970 has demonstrated a reduction in pro-inflammatory M1 macrophages as well as an increase in the concentration of M2 immunoregulatory macrophages. This is evidence of the dual mechanism of action and is particularly important as inflammatory macrophages are thought to play a key role in a number of inflammatory diseases, including RA.



Source: Toledo Roundtable Presentation

VALUATION

We model commercialization of filgotinib rheumatoid arthritis (RA) in the EU and Japan in 4Q20, and in the US in 2022 with a 50% risk adjustment, in inflammatory bowel disease (IBD) in 2022 with a 30% risk adjustment, in psoriatic arthritis (PsA) and ankylosing spondylitis (AS) in 2023, and in uveitis in 2025 with a 70% risk adjustment. We also factor ziritaxestat in 2023 with a 30% risk adjustment. A 30% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of $170.00**.**

Exhibit 54. Free Cash Flow Model.

Average	170

Price Target	155
Year	2021

DCF Valuation Using FCF (mln):

	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026
units ('000)	**2017A**	**2018A**	**2019A**	**2020E**	**2021E**	**2022E**	**2023E**	**2024E**	**2025E**	**2026E**
EBIT	(136,299)	(34,467)	177,070	(501,829)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
Tax Rate	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
EBIT (1-t)	(136,299)	(34,467)	177,070	(501,829)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
CapEx	-	(12,263)	(26,414)	(33,179)	(34,838)	(36,580)	(38,409)	(40,330)	(42,346)	(44,463)
Depreciation	-	5,996	14,689	20,563	21,591	22,670	23,804	24,994	26,244	27,556
Change in NWC										
FCF	(136,299)	(40,734)	165,344	(514,445)	(808,557)	(393,930)	(337,841)	288,196	816,667	1,391,692
PV of FCF	(238,389)	(61,951)	218,667	(591,612)	(808,557)	(342,548)	(255,456)	189,494	466,932	691,917

Discount Rate	15%
Long Term Growth Rate	1%
Terminal Cash Flow	10,040,063
Terminal Value YE2028	4,991,686
NPV	4,933,467
Net Cash 2021E	5,380,941
Shares out ('000)	66,481
NPV Per Share	155

2026E

Source: Maxim estimates

Exhibit 55. Discounted-EPS Model.

Current Year	2021
Year of EPS	2026
Earnings Multiple	10
Discount Factor	15%
Selected Year EPS	21.19
Net Cash Per Share (2020E)	80.94
NPV	186

Source: Maxim estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant						
	186.28	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**
Earnings Multiple	**0**	81.03	81.03	81.03	81.03	81.03	81.03
	5	151.89	137.18	125.99	117.37	110.66	105.38
	10	222.75	193.34	170.96	153.72	140.30	129.74
	15	293.62	249.50	215.92	190.07	169.93	154.10
	20	364.48	305.66	260.89	226.41	199.57	178.46
	25	435.34	361.81	305.85	262.76	229.21	202.82
	30	506.21	417.97	350.82	299.11	258.84	227.18
	35	577.07	474.13	395.79	335.45	288.48	251.54

Exhibit 56. Sum-of-the-Parts Model.

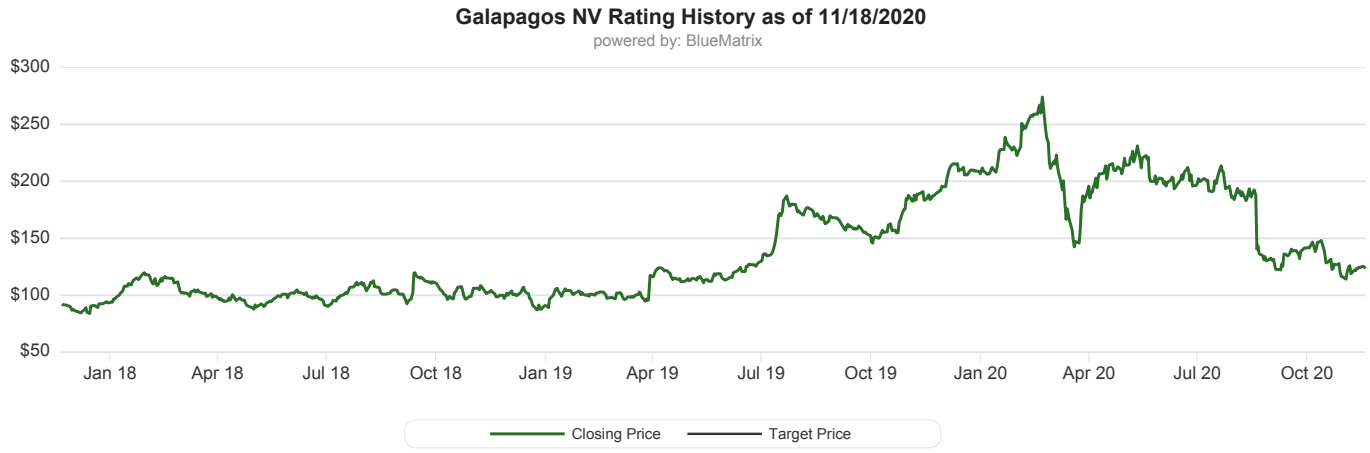
	LT Gr	Discount Rate	Yrs to Mkt	% Success	Peak Sales (MM's)	Term Value
Filgotinib - RA	1%	15%	1	40%	$925	$6,607
NPV						$19
Filgotinib - IBD	1%	15%	1	50%	$968	$6,915
NPV						$24.58
Filgotinib - Other	1%	15%	2	50%	$217	$1,551
NPV						$4.79
GLPG 1690 - IPF	1%	15%	2	50%	$1,621	$11,579
NPV						$36
Pipeline	1%	30%	5	25%	$2,500	$8,621
NPV						$5
Net Cash Per Share (2020E)						$5,381
Per Share						$80
Net Margin						55%
MM Shrs OS (2026E)						67
Total						$169

Source: Maxim estimates

.: YE December 31	2017A	2018A	2019A	1Q20A	2Q20A	3Q20A	4Q20E	2020E	2021E	2022E	2023E	2024E	2025E	2026E
Revenue:	-													
	-													
Filgotinib Sales (EU) - Rheumatoid Arthritis	-				-	-	5,710	5,710	110,889	206,016	257,207	285,438	315,530	347,588
Filgotinib Sales (EU) - Inflammatory Bowel Disease	-			-	-	-	-	-	-	15,811	75,848	155,195	190,002	226,332
Filgotinib Sales (EU) - Other Inflammatory Conditions	-			-	-	-	-	-	-	-	11,651	23,839	39,364	46,984
GLPG1690 Sales (EU) - Idiopathic Pulmonary Fibrosis										-	84,255	344,791	529,113	766,861
	-													
Net revenue	-	-	-	-	-	-	5,710	5,710	110,889	221,827	428,961	809,263	1,074,009	1,387,765
Collaborative revenue:	-													
Revenues	149,963	340,826	997,082	115,844	122,248	155,543		393,635		325,000				
Other Income	34,019	34,231	60,068	10,317	16,590	14,397		41,304	-	-	-	-	-	-
Milestones								-	-					
Filgotinib Royalties (US + Japan) - Rheumatoid Arthritis	-	-	-		-	-	1,905	1,905	27,337	84,749	144,474	217,127	303,813	378,663
Filgotinib Royalties (US + Japan) - Inflammatory Bowel Disease				-	-	-	-	-	-	20,490	108,293	242,107	321,617	413,242
Filgotinib Royalties (US + Japan) - Other Inflammatory Conditions				-	-	-	-	-	-	-	25,548	54,831	92,575	110,548
GLPG1690 Royalties (US) - Idiopathic Pulmonary Fibrosis										-	56,944	122,705	236,081	372,216
	-	-	-					-	-	-	-	-	-	-
Total Collaborative Revenue	**183,982**	**375,057**	**1,057,150**	**126,161**	**138,838**	**169,940**	**1,905**	**436,843**	**27,337**	**430,238**	**335,258**	**636,769**	**954,086**	**1,274,668**
Total Revenue	**183,982**	**375,057**	**1,057,150**	**126,161**	**138,838**	**169,940**	**7,615**	**442,553**	**138,226**	**652,065**	**764,219**	**1,446,033**	**2,028,095**	**2,662,434**
Gross Margins:														
Cost of Goods Sold				-	-	-	1,142	1,142	19,960	33,274	51,475	80,926	107,401	138,777
%Gross Margin							80%	80%	82%	85%	88%	90%	90%	90%
Gross Profit	183,982	375,057	1,057,150	126,161	138,838	169,940	6,473	441,411	118,266	618,791	712,744	1,365,106	1,920,694	2,523,657
Operating Expenses:														
Research and Development	257,832	380,993	504,238	137,780	175,955	156,063	157,624	627,422	658,793	671,969	685,408	699,117	713,099	727,361
%R&D														
General and Administrative	28,810	42,045	86,967	29,384	44,454	31,775	36,541	142,155	149,263	179,115	188,071	191,832	195,669	199,582
%G&A														
Sales and Marketing	3,308	4,892	29,001	11,606	20,161	20,281	23,323	75,371	105,520	147,728	162,501	170,626	179,157	188,115
%S&M														
Operating Expenses	289,950	427,929	620,206	178,771	240,570	208,119	218,630	846,090	933,536	1,032,086	1,087,455	1,142,501	1,195,326	1,253,835
Operating Income (Loss)	(105,968)	(52,872)	436,945	(52,610)	(101,733)	(38,179)	(211,015)	(403,537)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
Fair value remeasurement	-	-	(214,340)	(24,224)	(695)	15,379		(9,540)	-	-	-	-	-	-
Other Financial Income	5,755	21,635	25,349	46,872	(30,013)	(238)		16,620	-	-	-	-	-	-
Other Financial expenses	(36,087)	(3,230)	(70,884)	(29,350)	(2,869)	(73,153)		(105,372)	-	-	-	-	-	-
	-	-	-					-	-	-	-	-	-	-
	-	-	-					-	-	-	-	-	-	-
	-	-	-					-	-	-	-	-	-	-
Total Other Income	**-**	**18,406**	**(259,875)**	**(6,702)**	**(33,577)**	**(58,012)**	**-**	**(98,292)**	**-**	**-**	**-**	**-**	**-**	**-**
Pretax Income	(136,299)	(34,467)	177,070	(59,313)	(135,309)	(96,191)	(211,015)	(501,829)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
Taxes on income	234	59	253	396	440	457	-	1,293	-	-	-	-	-	-
Tax Rate														
GAAP Net Income (Loss)	(136,533)	(34,526)	176,817	(59,709)	(135,750)	(96,648)	(211,015)	(503,122)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
Total comprehensive loss	(136,533)	(34,526)	176,817	(59,709)	(135,750)	(96,648)	(211,015)	(503,122)	(795,309)	(380,021)	(323,236)	303,532	832,769	1,408,599
GAAP-EPS	(2.76)	(0.66)	3.07	(0.92)	(2.09)	(1.49)	(3.25)	(7.75)	(12.20)	(5.81)	(4.92)	4.60	12.58	21.19
GAAP-EPS (Dil)	(2.76)	(0.66)	2.94	(0.92)	(2.09)	(1.49)	(3.25)	(7.75)	(12.20)	(5.81)	(4.92)	4.60	12.58	21.19
Wgtd Avg Shrs (Bas) - '000s	49,479	52,113	57,633	64,873	64,873	64,938	65,003	64,922	65,166	65,427	65,689	65,952	66,216	66,481
Wgtd Avg Shrs (Dil) - '000s	49,479	52,113	60,179	64,873	64,873	64,938	65,003	64,922	65,166	65,427	65,689	65,952	66,216	66,481

Source: Company reports and Maxim

DISCLOSURES



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Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**81%**	**52%**
Hold	Fundamental metrics are currently at, or approaching, industry averages. Therefore, we expect this stock to neither outperform nor underperform its relevant index over the next 12 months.	**19%**	**50%**
Sell	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months.	**0%**	**0%**
	**See valuation section for company specific relevant indices*		

I, Jason McCarthy, Ph.D., attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

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Maxim Group makes a market in Galapagos NV

Maxim Group expects to receive or intends to seek compensation for investment banking services from Galapagos NV in the next 3 months.

GLPG: For Galapagos, we use the BTK (NYSE Biotechnology Index) as the relevant index.

Valuation Methods

GLPG: We model commercialization of filgotinib rheumatoid arthritis (RA) in the EU and Japan in 4Q20, and in the US in 2022 with a 50% risk adjustment, in inflammatory bowel disease (IBD) in 2022 with a 30% risk adjustment, in psoriatic arthritis (PsA) and ankylosing spondylitis (AS) in 2023, and in uveitis in 2025 with a 70% risk adjustment. We also factor ziritaxestat in 2023 with a 30% risk adjustment. A 30% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target.

Price Target and Investment Risks

GLPG: Aside from general market and other economic risks, risks particular to our price target and rating for Galapagos NV. include:(1) the regulatory and clinical risk associated with product development; (2); the rate and degree of progress of product development;(3) the rate of regulatory approval and timelines to potential commercialization of products; (4) the level of success achieved in clinical trials; (5) the requirements for marketing

authorization from regulatory bodies in the United States and other countries; (6) the liquidity and market volatility of the company's equity securities; (7) regulatory and manufacturing requirements and uncertainties; (8) product and technology developments by competitors, potentially with more resources and commercial infrastructure; (9) inability, if product(s) is approved to gain adequate market share;(10) impact of comprehensive tax reform in the US and Ex-US tax policy; (11) delays related to COVID-19 could impact the company's ability operate and conduct clinical trials; (12) failure of third-parties to meet contractual obligations, potentially impacting drug development; (13) Gilead is responsible for commercialization in the US as well as other regions, which limits the influence which Galapagos has on commercialization in the largest pharmaceutical market; (14) the result of the upcoming Type A meeting will likely determine the path forward for filgotinib in the US, if the 200mg dose does not move forward, Gilead is unlikely to commercialize the product in the US outside of inflammatory bowel disease, which would limit the commercial opportunity.

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Risk ratings take into account both fundamental criteria and price volatility.

Speculative – Fundamental Criteria: This is a risk rating assigned to early-stage companies with minimal to no revenues, lack of earnings, balance sheet concerns, and/or a short operating history. Accordingly, fundamental risk is expected to be significantly above the industry. Price Volatility: Because of the inherent fundamental criteria of the companies falling within this risk category, the price volatility is expected to be significant with the possibility that the investment could eventually be worthless. Speculative stocks may not be suitable for a significant class of individual investors.

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