March 21, 2019

OUTPERFORM

Reason for report:

PROPRIETARY INSIGHTS

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GALAPAGOS NV

Diversity in the I&I Archipelago – Reiterating Outperform, $140 PT

• **Bottom Line:** GLPG's opportunities with lead drugs filgotinib, a highly selective JAK1 inhibitor, and GLPG1690, a first-in-class ATX inhibitor, were enabled by their assembly of elite European scientific talent that has developed a world-class discovery platform coupled with in-house chemistry. Since the $725M licensing deal with collaborative partner GILD in 2015, filgotinib has advanced into ongoing Phase 3 trials in rheumatoid arthritis (RA), Crohn's disease (CD) and ulcerative colitis (UC), and Phase 2 trials in eight additional inflammatory diseases. We are very positive on filgotinib ahead of the highly anticipated readouts for the Phase 3 FINCH1 and FINCH3 trials for filgotinib to treat RA. We project the Phase 3 readouts create $10 upside versus $20 downside risk to our price target but assume a 75% probability of regulatory success. Meanwhile, GLPG's intermediate future centers on idiopathic pulmonary fibrosis (IPF) with GLPG1690 recently entering twin Phase 3 trials to treat IPF, where we see a high potential for it to become the standard-of-care if approved. Further down the pipeline, Phase 2 candidate GLPG1972 provides a high risk-reward opportunity in osteoarthritis while the Toledo program represents GLPG's next step in immunology and inflammation (I&I) with multiple molecules expected to enter clinical trials over the next two years. We reiterate our Outperform rating and 12-month price target of $140 per ADS.

Key Stats: **(NASDAQ: GLPG)**

Sector:	**Biopharma / Immunology & Metabolism**
S&P 500 Health Care Index:	**1,064.97**
Price :	**$103.24**
Price Target:	$140.00
Methodology:	SOTP with WACC-calculated 11.9% discount rate and a 2% terminal growth rate to the discounted cash flow value of each asset. DCF values were adjusted by asset specific probability of regulatory success.
52 Week High:	$122.28
52 Week Low:	$85.00
Shares Outstanding (mil):	54.4
Market Capitalization (mil):	$5,616.3
Book Value/Share:	$0.00
Cash Per Share:	$24.78
Dividend (ann):	€0.00
Dividend Yield:	0.0%

Completion: March 21, 2019, 6:59AM EDT.
Distribution: March 21, 2019, 6:59AM EDT.

Table of Contents:

Dec Yr	1Q	2Q	3Q	4Q	FY Rev	1Q	2Q	3Q	4Q	FY EPS	P/E
2018A	**€44.8**	**€57.0**	**€103.2**	**€112.8**	**€317.8**	**(€0.73)**	**(€0.42)**	**€0.29**	**€0.28**	**(€0.56)**	**NM**
2019E - New	**€36.2**	**€36.3**	**€36.3**	**€45.1**	**€153.8**	**(€1.24)**	**(€1.28)**	**(€1.32)**	**(€1.18)**	**(€5.02)**	**NM**
2019E - Old	€36.2	€36.3	€36.3	€45.1	€153.8	(€1.02)	(€1.01)	(€1.01)	(€0.85)	(€3.90)	NM
2020E - New	**--**	**--**	**--**	**--**	**€203.5**	**--**	**--**	**--**	**--**	**(€4.54)**	**NM**
2020E - Old	--	--	--	--	€198.6	--	--	--	--	(€3.18)	NM

Source: Company Information and SVB Leerink LLC Research.
Revs in €MM
EPS diluted non-GAAP

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Diversity in the I&I Archipelago – Reiterating Outperform, $140 PT

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Chapter Summary

Chapter 1: Filgotinib

- Highly selective JAK1 inhibitor
- Partnered with Gilead in $2.1B licensing agreement
- Currently in three Phase 3 trials and eight Phase 2 trials to treat a suite of inflammatory diseases

Indication	Stage
Rheumatoid arthritis (RA)	Phase 3
Ulcerative colitis (UC)	Phase 3
Crohn's disease (CD)	Phase 3
Small bowel CD	Phase 2
Fistulizing CD	Phase 2
Sjogren's disease	Phase 2
Ankylosing spondylitis	Phase 2
Psoriatic arthritis (PA)	Phase 2
Cutaneous lupus (CL)	Phase 2
Lupus nephropathy (LN)	Phase 2
Uveitis	Phase 2

Chapter 2: Idiopathic Pulmonary Fibrosis

- Positioned to be a leader in IPF
- GLPG1690: First-in-class autotaxin (ATX) inhibitor
- Currently in Phase 3 trial (ISABELLA)
- Orphan status in the U.S and E.U.
- GLPG1205: G-protein receptor 84 (GPR84) inhibitor
- Currently in Phase 2 trial (PINTA)
- Previously shown to be well tolerated in UC

Chapter 3: Osteoarthritis

- GLPG1972: ADAMTS-5 inhibitor
- Partnered with Servier in €290M licensing agreement
- Currently in Phase 2 study (ROCELLA)
- Fast track designation with the FDA

Chapter 4: Discovery

- Proprietary discovery platform
- Mor106 exemplifies the partnership potential for this platform

Sources: Company reports; SVB Leerink Research

Strong push into IPF Creates the Path Forward for GLPG Past Filgotinib

Ticker	SVB Leerink Rating	SVB Leerink PT	Date	Close	ADSs Outstanding (M)	52-week Low	52-week High	Market Cap (B)	Cash/share	Cash & Equiv. (M)
GLPG	Outperform	$140	3/19/2019	$101.06	54.47	$85.00	$122.28	$5.5	€ 23.70	€ 1,291

Company Overview

- **Galapagos at a Glance:**
  - Belgian based company founded in 1999 based on a proprietary drug discovery platform
  - Their platform has produced drug candidates targeting IPF, inflammatory diseases, OA, atopic dermatitis, and opened up partnerships with leading pharmaceutical companies.
  - Trades on the Euronext (GLPG) and as ADRs on the NASDAQ exchange (GLPG)
- **Financials:**
  - €1.3B in cash as of 4Q18
  - Market cap €4.9B ($5.5B)
  - €325M projected 12-month cash burn
  - Projected cash runway into 2023
- **Valuation Methodology:**
  - Sum of the parts valuation applying an 11.9% WACC calculated discount rate and 2% terminal growth rate.
  - Each drug's constituent values are determined by a probability weighted scenario analysis based our independent asset profiles

Investment Thesis

- Filgotinib's superior safety profile versus other JAKi's makes it a potentially best-in-class JAKi that has an opportunity to compete with anti-TNF agents in RA and IBD.
- We believe GLPG has a potential best-in-care drug in GLPG1690 that has the potential to become the standard of care in IPF treatment.
- With limited effective treatments in IPF, GLPG1690 carries blockbuster potential with a projected 2022 market launch.
- While we view gaining approval to treat OA highly unlikely given the history of clinical efforts, we cannot ignore the massive opportunity should GLPG1972 gain regulatory approval.
- We expect GLPG's proprietary discovery platform to continue to produce uniquely profiled drugs that will keep the doors open to partnership opportunities

Expectations

- **Value creation:**
  - With Phase 3 trials being the key near term driver, we anticipate PoC trials and execution updates regarding trial status to create steadier upside value
  - Advancement of new programs such as the Toledo program will continue to expand GLPG's pipeline
- **Likelihood of technical, regulatory, access, and commercial success**
  - We assign probabilities of regulatory approval of 40.7%, and 68% to GLPG1690 and Filgotinib, respectively. These drugs shift GLPG towards a meaningful commercial market profile
- **Risks:**
  - Regulatory and commercial path for filgotinib is under the control of a collaborative partner, Gilead
  - Filgotinib fails to be differentiated against other JAK inhibitors
  - Path forward for GLPG1690 as combination therapy in IPF leaves the backdoor exposed to monotherapy pursuit by competitors

Sources: Company filings; SVB Leerink research

Pursuing large markets with drug pipeline

	Pre-IND	Phase 1	Phase 2	Phase 3	NDA	Market
Filgotinib	Rheumatoid arthritis, Crohn's disease, Ulcerative colitis					2020 & 2021
Idiopathic Pulmonary Fibrosis	GLPG1690 & GLPG1205					2022
Osteoarthritis	GLPG1972					2025
Toledo	Inflammatory disease					

Drug	Indication	Event	Timing	Importance
Filgotinib	RA	FINCH1 and FINCH3 Phase 3 trials topline results	1Q19	High
MOR106	Atopic dermatitis	Complete Phase 2 recruitment	1H19	Low
Filgotinib	Sjögren's disease	Phase 2 proof of concept topline results	2H19	Medium
Filgotinib	CLE	Phase 2 proof of concept topline results	2H19	Medium
Filgotinib	Psoriatic arthritis	Initiate Phase 3	2H19	Medium
Filgotinib	RA	File NDA	4Q19	Medium
GLPG1205	IPF	Complete Phase 2 recruitment	2H19	Low
GLPG1972	OA	Complete Phase 2b recruitment	2H19	Low
GLPG3970	Inflammatory diseases	Initiate Phase 1	2H19	Low
GLPG3312	IBD	Initiate Phase 2 proof-of-concept	2H19	Low

Source: Company presentations, SVB Leerink *research*

Chapter 1

Filgotinib- Selective Oral JAK1 inhibitor for Inflammatory Diseases

Chapter Overview

Prospectus

- Filgotinib is a highly selective JAK1 inhibitor being developed to treat inflammatory diseases
- Filgotinib yields an active metabolite that is also highly JAK1 selective
- Pipeline in a drug
- Improved safety profile vs other JAK inhibitors
- Efficacy and safety profile more aligned with TNF inhibitors than JAK inhibitors

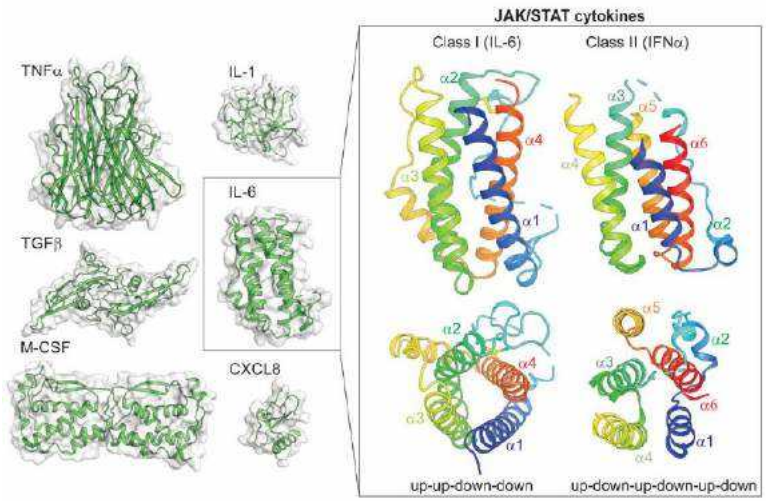
Implications

- High selectivity for JAK1 vs JAK2, in particular provides better opportunity to increase effective dose while minimizing safety concerns
- The active metabolite serves to increase the overall potency of filgotinib towards JAK1 inhibition
- With development proceeding in eleven indications spread across three Phase 3 trials (RA, CD and UC) and eight Phase 2 trials, filgotinib offers multiple shots on goal
- With a better safety profile to date, particularly related to reduced incidences of serious infections and thromboembolic events, filgotinib avoids many of the concerns surrounding the use of other JAK inhibitors
- The average efficacy matched with a better safety profile enables targeting of earlier lines of treatment, placing it in competition with TNF inhibitors rather than with other JAK inhibitors

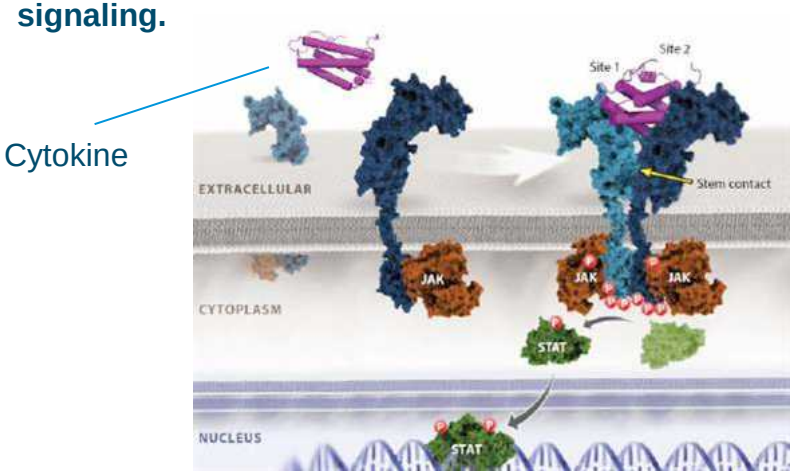
Source: SVB Leerink Research

JAK-STAT signaling at the heart of the immune system

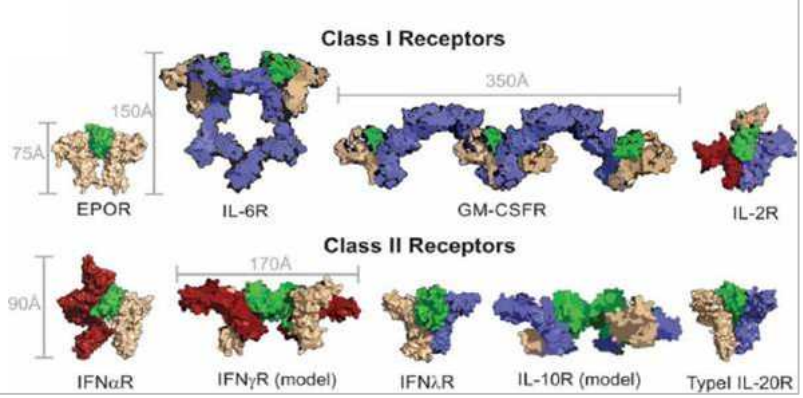
Structures of members of the TNFα-family, TGFβ-family, IL-1-like cytokines, chemokines (CXCL8), cytokines that signal through receptor tyrosine-kinases (M-CSF) or the JAK/STAT pathway (IL-6)



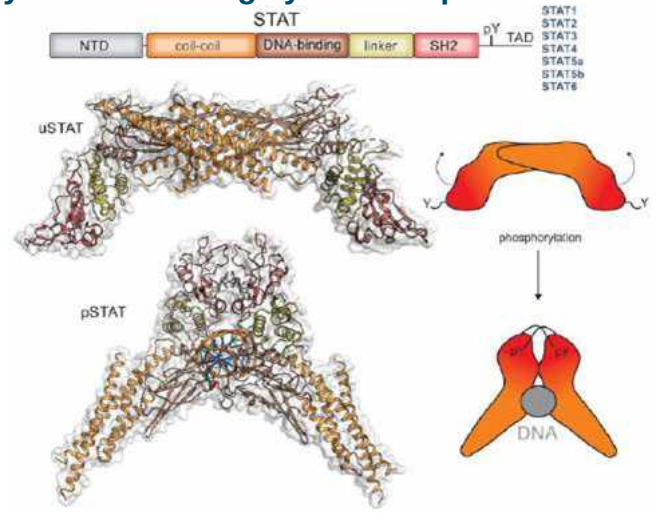
Cytokine receptor–ligand engagement, dimerization, and signaling.



Structures and models of a diverse range of cytokine:receptor complexes

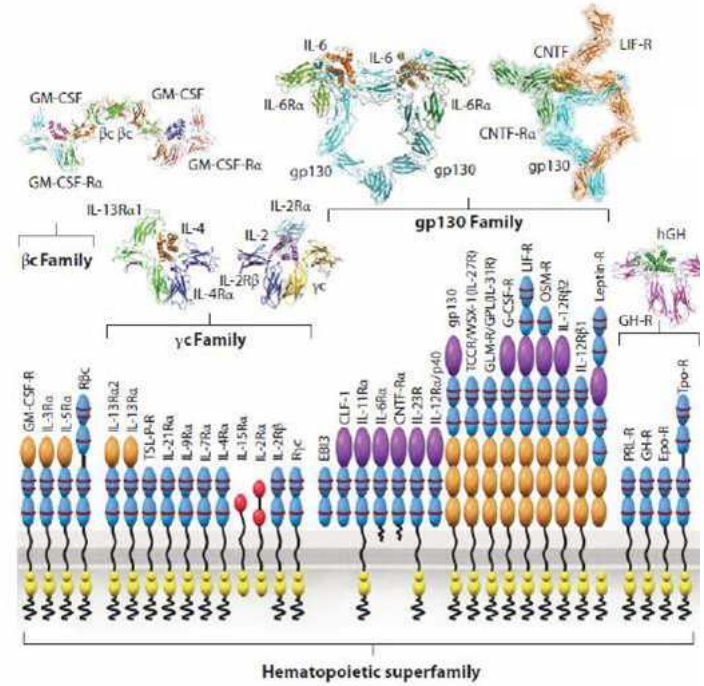


Signal Transducers and Activators of Transcription (STATs) are a family of latent transcription factors that are activated by phosphorylation following cytokine exposure



Source: Kevin 2017, O'Shea 2018, Jones JMedChem 2016, leonard Nature Chem Bio 2016, Smith Nature Chem Bio 2016, Shi J Med Chem 2018, Thoma J Med Chem 2011, Kempson Bioo Med Chem Letters 2017, Clark J Med Chem 2014, kim J Med Chem 2015, Schenkel J Med Chem 2011, Thorarensen Chem Bio 2014, Soth J Med Chem 2012, Winthrop Nature Rheum 2017, Tan J Med Chem 2015, Menet J Med Chem 2014, Schwartz NRDD 2017

JAK STAT--- Class I Cytokines: Defense, Offense, Growth and Repair



IL-6 family

IL-6	Pleiotropic, haematopoiesis, acute phase response, lymphoid differentiation
LIF	Pleiotropic, blastocyst implantation, bone remodeling, CNS
CNTF	Neuronal growth factor
CT1	Cardiac myocytes growth factor
CLC	Neurological growth factor
OSM	Pleiotropic, bone formation
IL-31	Inflammatory, cell-mediated immunity
NP	Neural growth factor

IL-2 family

IL-2	Immune response, T-cell differentiation
IL-4	T_H2 differentiation
IL-7	T-, B-cell growth factor
IL-9	Pleiotropic, Stimulates, T-, B- and NK cells
IL-15	Stimulates T- and NK-cells
IL-21	Stimulates, T-, B- and NK cells

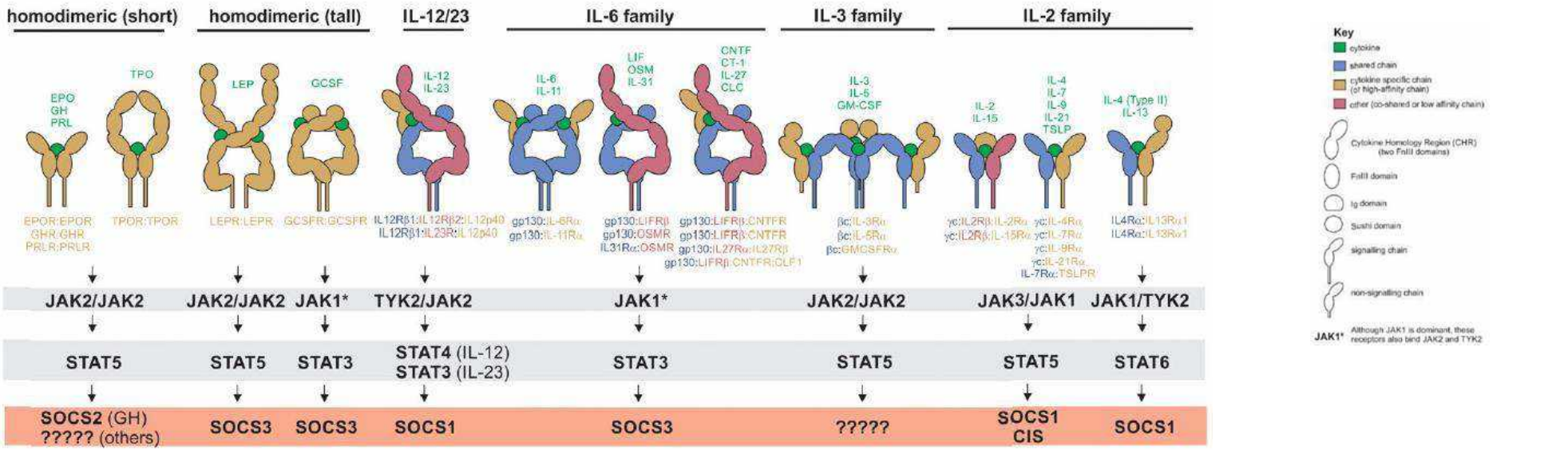
IL-3 family

IL-3	Multi-lineage haematopoietic growth factor
IL-5	B-cell development, eosinophils
GM-CSF	Multi-lineage haematopoietic growth factor, especially monocytes, neutrophils, eosinophils and basophils

G-CSF	Stimulates granulocyte production, mobilises stem cells
EPO	Stimulates formation of erthrocytes
TPO	Stimulates formation of megakaryocytes/platelets
GH	Growth
PRL	Milk production
LEP	Regulates appetite

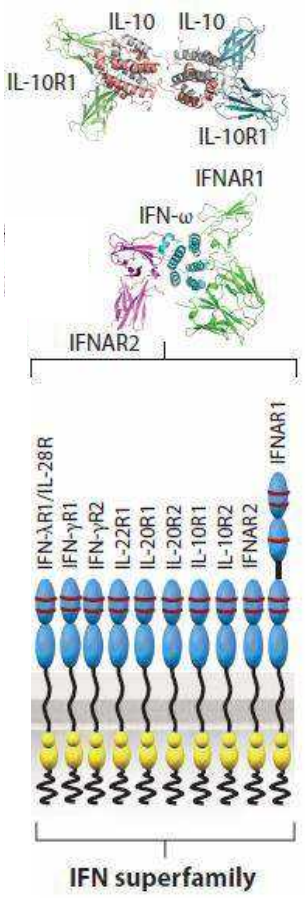
Others

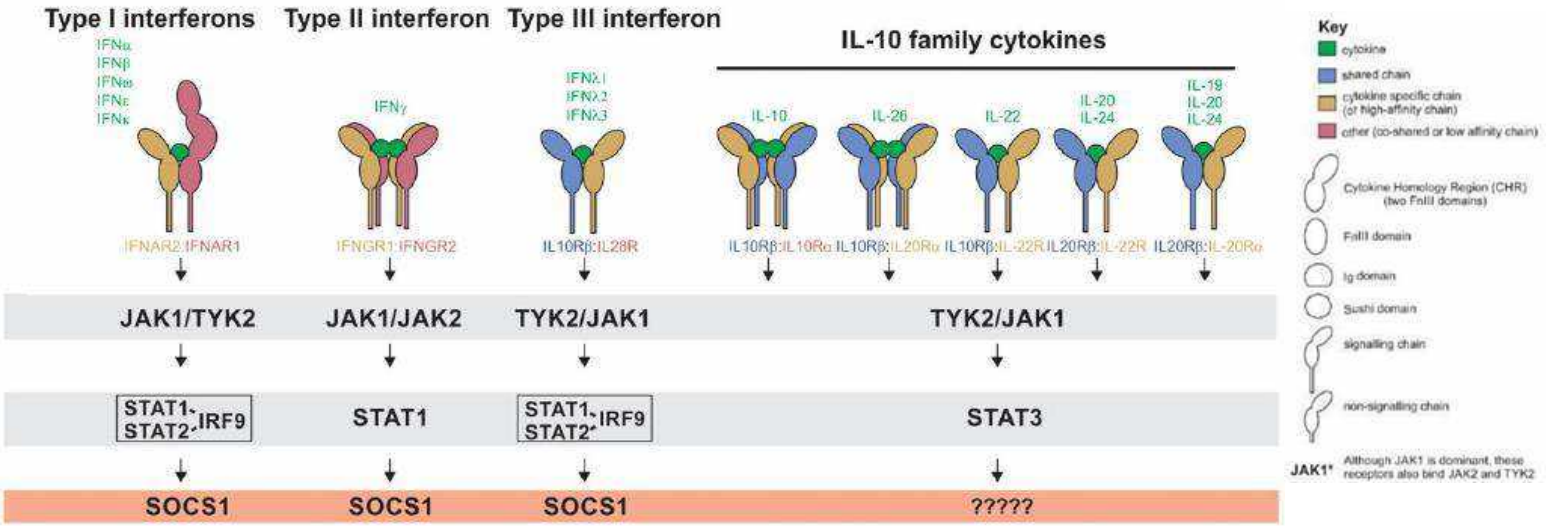
IL-12	[illegible]
IL-13	[illegible]
IL-23	[illegible]
TSLP	[illegible]



Source: Kevin 2017, O'Shea 2018, Jones JMedChem 2016, leonard Nature Chem Bio 2016, Smith Nature Chem Bio 2016, Shi J Med Chem 2018, Thoma J Med Chem 2011, Kempson Bioo Med Chem Letters 2017, Clark J Med Chem 2014, kim J Med Chem 2015, Schenkel J Med Chem 2011, Thorarensen Chem Bio 2014, Soth J Med Chem 2012, Winthrop Nature Rheum 2017, Tan J Med Chem 2015, Menet J Med Chem 2014, Schwartz NRDD 2017

JAK STAT --- Class II Cytokines: Defense against the elements





Class II cytokines		
Type I interferon		
IFNα	Interferon alpha (23 subtypes)	Anti-viral, secreted by lymphocytes, fibroblasts and monocytes
IFNβ	Interferon beta	Anti-viral, ubiquitously expressed
IFNε	Interferon epsilon	Anti-viral, expressed in female reproductive tract
IFNκ	Interferon kappa	Anti-viral, expressed by keratinocytes
IFNω	Interferon omega	Anti-viral, secreted by leukocytes
Type II interferon		
IFNγ	Interferon gamma	Pro-inflammatory, secreted by T- and NK-cells, activates macrophages/monocytes
Type III interferon		
IFNλ1	Interferon lambda1	Anti-viral, similar to type I but acts on fewer cell-types
IFNλ2	Interferon lambda2	Anti-viral, similar to type I but acts on fewer cell-types
IFNλ3	Interferon lambda3	Anti-viral, similar to type I but acts on fewer cell-types
IL-10 family		
IL-10	Interleukin-10	Anti-inflammatory, inhibits macrophage activation
IL-19	Interleukin-19	Inflammatory, acts on dermal cells
IL-20	Interleukin-20	Inflammatory, acts on dermal cells
IL-22	Interleukin-22	Inflammatory, secreted by Th1 cells, acts on dermal cells
IL-24	Interleukin-24	Inflammatory, acts on dermal cells
IL-26	Interleukin-26	Antimicrobial, T_H17 cytokine

Source: Kevin 2017, O'Shea 2018, Jones JMedChem 2016, leonard Nature Chem Bio 2016, Smith Nature Chem Bio 2016, Shi J Med Chem 2018, Thoma J Med Chem 2011, Kempson Bioo Med Chem Letters 2017, Clark J Med Chem 2014, kim J Med Chem 2015, Schenkel J Med Chem 2011, Thorarensen Chem Bio 2014, Soth J Med Chem 2012, Winthrop Nature Rheum 2017, Tan J Med Chem 2015, Menet J Med Chem 2014, Schwartz NRDD 2017

Understanding the JAK selectivity: relying on one assay a big mistake

Published enzymatic and cellular IC50 (nM) from various sources

	Enzymatic IC50 (nM)			
	JAK-1	JAK-2	JAK-3	TYK2
Baricitinib	4	6.6	787	61
Decernotinib	112	619	74.4	10,000
Filgotinib	363	2400	10,000	2,600
Ruxolitinib	6.4	8.8	487	30.1
Tofacitinib	15.1	77.4	55	489
Tofacitinib	15	77	55	489
Peficitinib	4	5	1	5
Ruxolitinib	6	9	487	30
Baricitinib	4	7	787	61
Fedratinib	105	3	1002	405
BMS-911543	360	1	75	66
Decernotinib	112	619	74.4	10,000
Filgotinib	363	2400	10,000	2600
PF-04965842	29	803	10,000	1253
Itacitinib	43	120	2300	4700
Upadacitinib	2	68	280	12
VX-509	11	13	2	11
PF- 6647511 (Baricitinib)	4.5	7	601	50
CP-690550-10 (Tofacitinib)	15.1	77.4	55	489
PF-02384554	2.75	700	10,000	260
PF-06263313	10,000	10,000	40	10,000
PF-06651600	10,000	10,000	33.1	10,000

	Cellular IC50 (nM)- Human PBMC					
	pSTAT5 IL-15	pSTAT1 IL-6	pSTAT4 IL-12	pSTAT3 IL-23	pSTAT3 IFNα	pSTAT5 CD34
Baricitinib	259	21.1	149	81.9	28.7	87.8
Decernotinib	932	1,870	16,400	11,200	1,290	20,000
Filgotinib	2,140	918	13,362	10,123	1,500	13,200
Ruxolitinib	1,850	298	1,090	818	194	677
Tofacitinib	55.8	75.4	409	229	35	302

	Cellular IC50 (nM)- Mouse PBMC						
	pSTAT5 (J1/3) IL-15	pSTAT5 (J1/3) IL-2	pSTAT5 (J1/3) IL-21	pSTAT3 (J1/T2) IL-10	pSTAT3 (J1/T2) IFNα	pSTAT3 (J1/J2/J2) IL-27	pSTAT3 (J2/T2) IL-23
PF- 6647511 (Baricitinib)	12.6				3.5	3.5	26
CP-690550-10 (Tofacitinib)	15.3	15	21.3	42.1	9.38	8	125
PF-02384554	20.6			39.7	5.56	5.14	1050
PF-06263313	116	122	103	10,000	9640	12050	
PF-06651600	49.9		70.4	10,000	9000	12308	

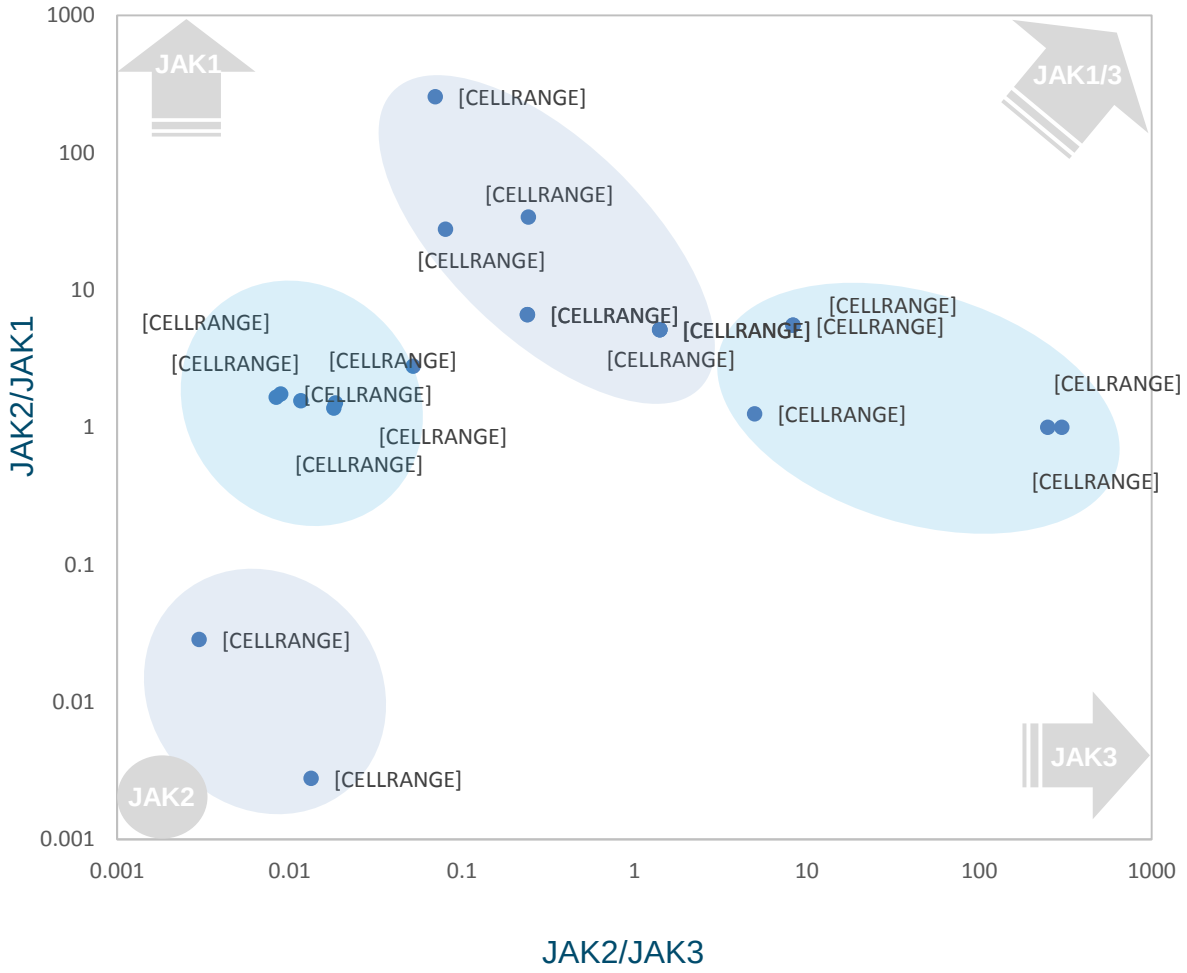
- In vitro enzymatic assays are very important to distinguish properties of inhibitors
- However, the logic of JAK activity is only apparent in vivo

Sources: Moodley et al. 2016; Clark et al. 2014; Lin et al 2010; Rompaey et al. 2013; Company reports

Understanding JAK selectivity

JAK selectivity with an eye for 1 and 3

Ratio of published IC50's across JAK inhibitors

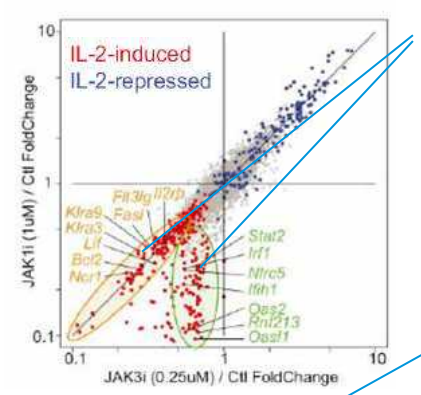


- Most approved or pipeline medicines hit multiple kinases, albeit with varying potencies
- While potencies vary it is very difficult to draw conclusions regarding clinical safety and efficacy
- Strategies have veered from expedience to highly selective inhibitors (which are making their way through clinic)
- Assays vary depending on properly using ATP at cellular Km's vs high ATP (lots of various IC50 values per molecule and misunderstanding of IC50's due to these disconnects)
- Full coverage of a kinase is great but sometimes a weaker profile provides a broader therapeutic window

Source: Kevin 2017, O'Shea 2018, Jones JMedChem 2016, leonard Nature Chem Bio 2016, Smith Nature Chem Bio 2016, Shi J Med Chem 2018, Thoma J Med Chem 2011, Kempson Bioo Med Chem Letters 2017, Clark J Med Chem 2014, kim J Med Chem 2015, Schenkel J Med Chem 2011, Thorarensen Chem Bio 2014, Soth J Med Chem 2012, Winthrop Nature Rheum 2017, Tan J Med Chem 2015, Menet J Med Chem 2014, Schwartz NRDD 2017

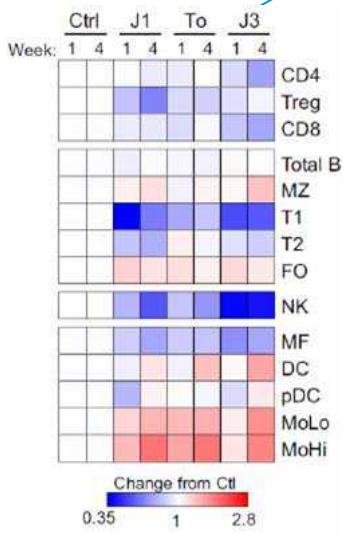
Understanding JAK selectivity: a network effect with nuances

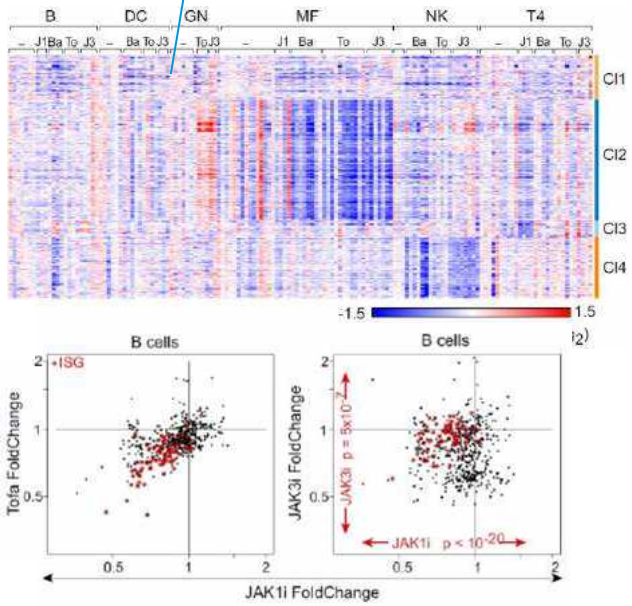
JAKi's have a profound impact on immunogenomics across cell types; nuances exist that will likely differentiate one inhibitor from another, which are unlikely to be identified in simple enzyme or cellular assays



Jaki's have both overlapping and selective gene program suppression

Broad reprogramming of immunocyte quantities with different JAKi's



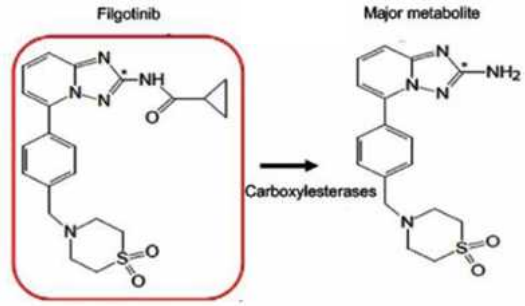


Implications of network effects of JAKi:

1. Whether on cells or genes, the effects are broad but subtle, overlapping between compounds, even compounds that target single JAK isoforms with high specificity
2. The signaling and transcriptional network adapts to repeated JAK blockade, with the drug's effect persisting even after the compound has cleared
3. When two JAK isoforms are involved in signals from a given cytokine, selectively blocking one or the other has a different impact on that cytokine's overall signature
4. TH1/17 and T cells are important, but innate mechanisms including NK cells, have a important contribution to observed effects of JAK inhibitors
5. Isoform-specific JAKi will not provide sharply delineated blockade of a specific pathway, but quantitative nuances of network-level effects, which may differentiate therapeutic windows relative to adverse events

Source: ACR 2018, EULAR 2018, O'Shea 2018, Jones JMedChem 2016, leonard Nature Chem Bio 2016, Smith Nature Chem Bio 2016, Shi J Med Chem 2018, Thoma J Med Chem 2011, Kempson Bioo Med Chem Letters 2017, Clark J Med Chem 2014, kim J Med Chem 2015, Schenkel J Med Chem 2011, Thorarensen Chem Bio 2014, Soth J Med Chem 2012, Winthrop Nature Rheum 2017, Tan J Med Chem 2015, Menet J Med Chem 2014, Schwartz NRDD 2017, Moodley PNAS 2016

Filgotinib- a highly selective Jak1 inhibitor being developed in collaboration with Gilead



IC_{50} (nM)	JAK1	JAK1/JAK3	JAK1/TYK2	JAK2	Fold-selectivity JAK1 vs JAK2
Filgotinib	629	1,789	1,127	17,453	27.7
Metabolite	11,917	19,626	15,423	>100,000	>8.4

DEALS DECEMBER 17, 2015 / 1:55 AM / 3 YEARS AGO

Belgium's Galapagos signs $2 billion deal with U.S. group Gilead

REUTERS 2 MIN READ

BRUSSELS (Reuters) - Belgian biotech company Galapagos (GLPG.AS) has signed a development deal for drugs targeting inflammatory diseases potentially worth more than $2 billion with U.S. group Gilead (GILD.O).

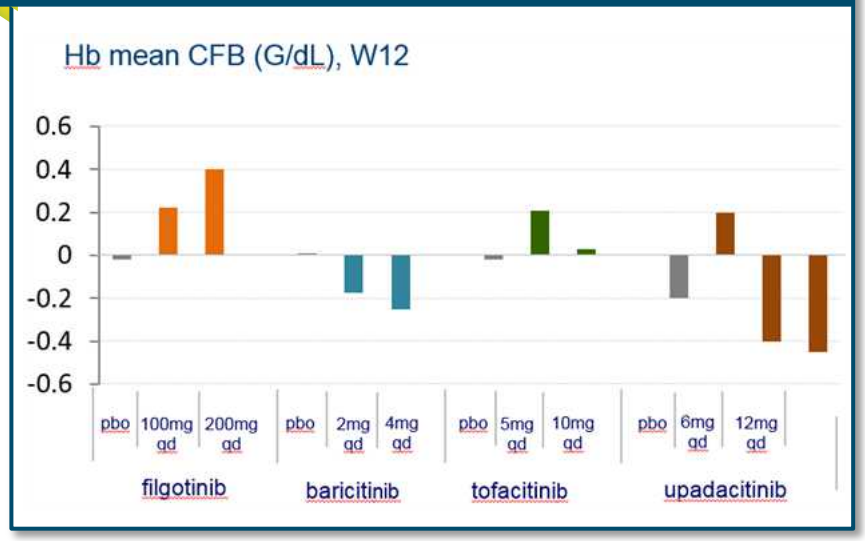
- Filgotinib was discovered in a screen of about 10,000 compounds.
- JAK1 specificity was optimized using post screen analysis of modifications to a core structure, resulting in the selection of filgotinib for further development
- Filgotinib yields a major metabolite that also exhibits high JAK1 specificity relative to the other JAK kinases, but at about 20-fold lower potency compared to filgotinib
- The presence of the metabolite serves to increase the overall per dose inhibitory effect

Agreement date	Upfront Cash Payment	Upfront equity investment	Total milestones	Commercial Royalties	Est. launch date (RA)	Est. launch date (IBD)
Dec. 2015	$300M	$425M	$1.35B	Tiered starting at 20%	2020	2022

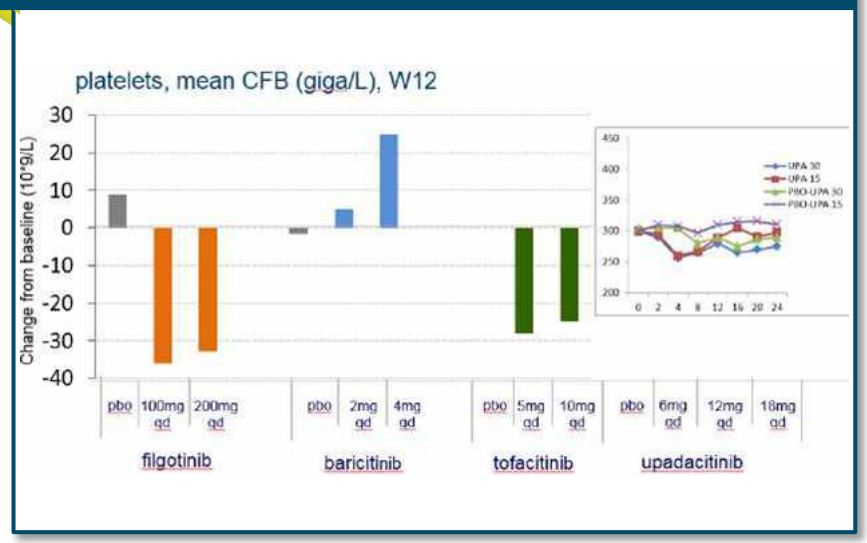
Source: Galien et al. 2013; Namour et al. 2016; Clark et al. 2014; Company reports.

Potential Markers for Reduced Bonemarrow-Related and Hematologic Toxicity

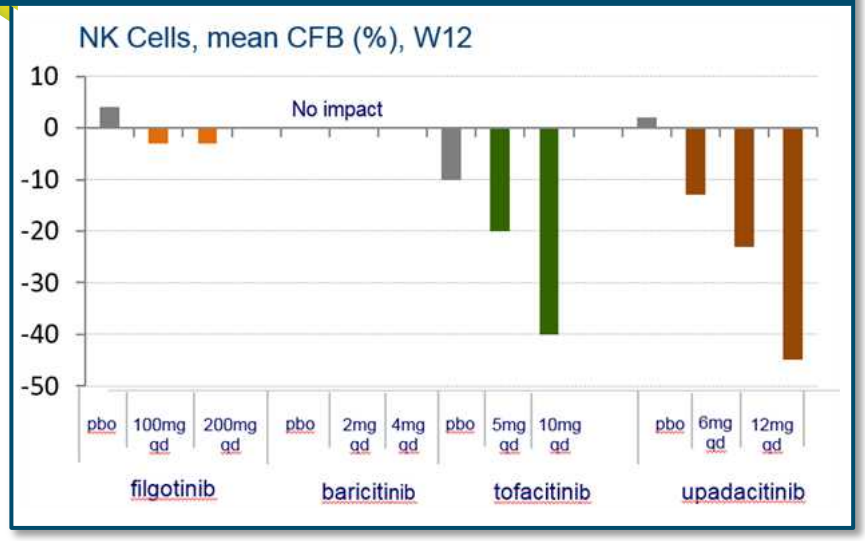
Filgotinib increased hemoglobin



Platelet reduction with filgotinib



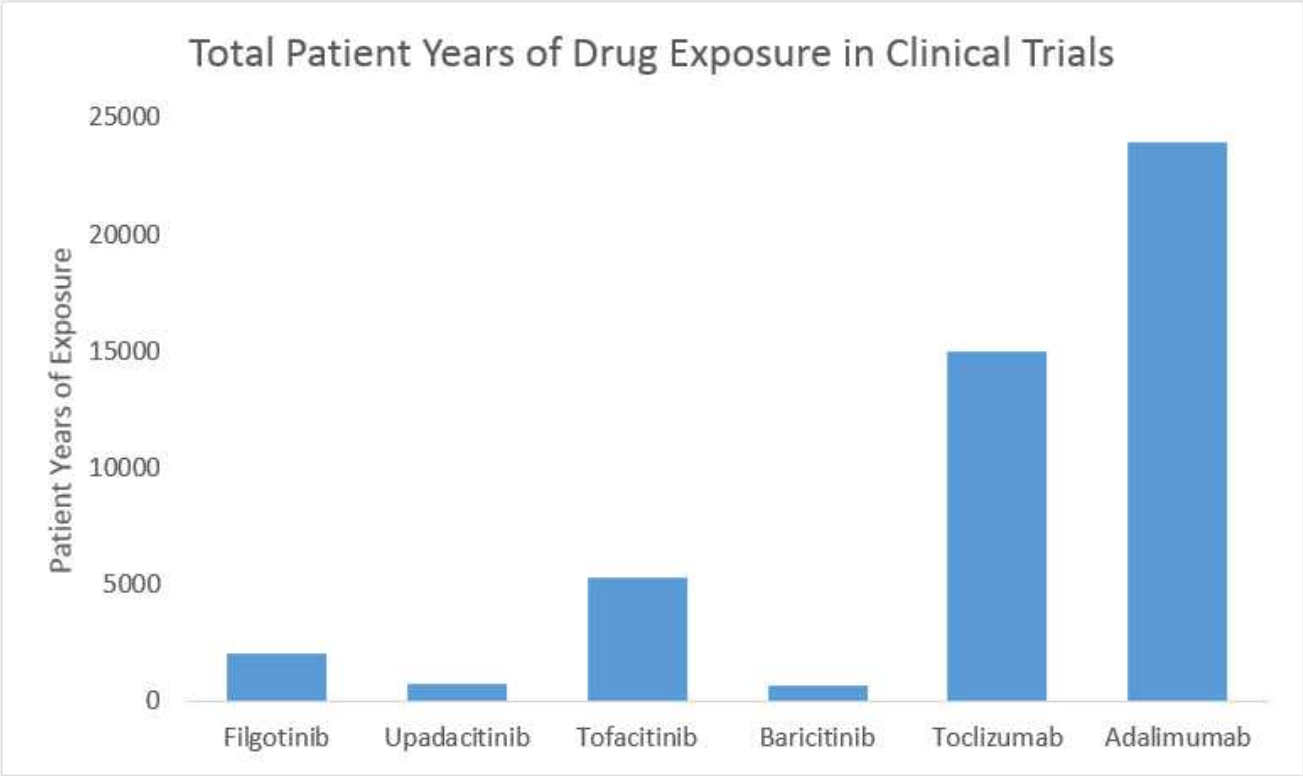
Filgotinib did not impact NK cell concentration



- We expect improved hematologic changes (hemoglobin, platelet and NK cell counts) will translate to reduced risk of thrombosis and anemia in patients
- Filgotinib has shown low impact on changes in NK cell concentration versus less selective JAK1 inhibitors and minor impact on lymphocyte concentrations, has thus far translated into improved infection rates compared to other JAK inhibitors.

Source: Company presentations; Mease et al. 2018; Genovese et al. 2017; Genovese 2017; Genovese 2012; Wollenhaupt 2017; Burmester 2011; SVB Leerink Research

Filgotinib's Safety Profile Differentiates it From Other JAKis and Biologics



Per 100 patient years	Filgotinib	Upadacitinib	Tofacitinib	Baricitinib	Toclizumab	Adalimumab
Serious Infection	1	2.3	2.4	2.9	4.5	4.6
Herpes zoster	1.5	3.7	3.8	3.2	ND	ND
DVT/PE	0.1	0.7	0.2	0.5	ND	ND
Deaths	0.2	0.3	0.6	0.3	0.6	0.8

Note: These numbers may change as new data becomes available

- Filgotinib has shown a better safety profile relative to other JAK inhibitors and biologics based on:
  - Reduced rate of serious infection
  - Reduced rate of infection Herpes zoster
  - Reduced deep vein thrombosis (DVT) and pulmonary embolism (PE)
  - And lower incidence of death
- These may be partially explained by the comparatively improved hematologic impact

Source: Company presentations; Mease et al. 2018; Genovese et al. 2017; Genovese 2017; Genovese 2012; Wollenhaupt 2017; Burmester 2011; SVB Leerink Research

Filgotinib- Pipeline in a drug

Indication	Pre-IND	Phase 1	Phase 2	Phase 3	NDA	Market (Est. Launch)
Rheumatoid arthritis (RA)	Phase 3 FINCH1, FINCH2, and FINCH3					2020
Ulcerative colitis (UC)	Phase 3 SELECTION1					2021
Crohn's disease (CD)	Phase 3 DIVERSITY1					2021
Psoriatic arthritis (PA)		Phase 2 EQUATOR				
Ankylosing spondylitis		Phase 2 TORTUGA				
Small bowel CD		Phase 2 SBCD				
Fistulizing CD		Phase 2 DIVERGENCE2				
Sjögren's disease		Phase 2				
Cutaneous lupus (CLE)		Phase 2				
Lupus nephropathy (LN)		Phase 2				
Uveitis		Phase 2 HUMBOLDT				

Sources: Company reports; SVB Leerink Research

RA Epidemiology and Pathology

Rheumatoid arthritis (RA) and its symptoms

- A chronic inflammatory disease characterized by swelling in the joints (synovitis) with synovial thickening, cartilage damage and bone erosion occurring as the disease progresses.
- Disease progression can lead to significant reduction in quality of life through destruction of synovial joints, disability, and increased mortality risk.
- Appearance of circulating anti-citrullinated protein antibodies (ACPA) which are involved in self-targeting of partially broken down (citrullinated) proteins that occurs during normal cell death
- Elevated levels of rheumatoid factor, a class of self targeting antibodies, and c-reactive protein, an indicator of systemic inflammation secreted by the liver
- Raised erythroid sedimentation rate (ESR)

Comorbidities

- Cardiovascular diseases such as myocardial infarction, cardiomyopathy, hypertension, peripheral vascular diseases
- Respiratory disease including chronic obstructive pulmonary disease (COPD), pleurisy, lung abscess, and pulmonary fibrosis
- Diabetes mellitus

	Total U.S. (range average)
Prevalence	~1.3 million (0.53% to 0.55%)
Female to male ratio	3 to 1
Mortality risk	54% increased risk vs non-RA population

Diagnosis Scoring System

Criteria	Description	Score
Morning Stiffness	Unexplained clinical synovitis in at least 1 joint	N/A
Joint Involvement	1 large joint	0
	2 to 10 large joints	1
	1 to 3 small joints	2
	4 to 10 small joints	3
	>10 joints, at least one small	5
Serology	Negatative RF and negative ACPA	0
	Low-positive RF or ACPA	2
	High-positive RF or ACPA	3
Acute phase reactants	Normal CRP and ESR	0
	Abnormal CRP and ESR	1
Duration of symptoms	<6 weeks	0
	≥6 weeks	1
Criteria score ≥6 out of 10 to be classified as RA		

Source: Aletaha et al. 2010; Gibosfsky et al. 2012; Graf et al. 2009; Ingegnoli et al. 2013; Sparks et al. 2016; Hunter et al. 2014; van den Hoek et al. 2017; Singh et al. 2015.