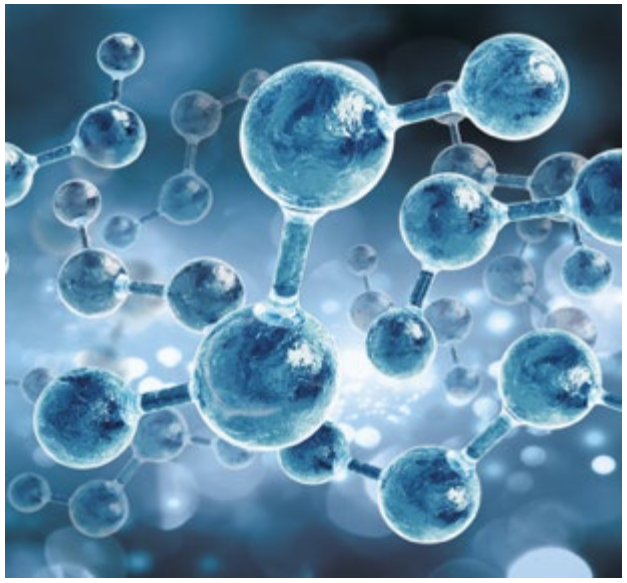
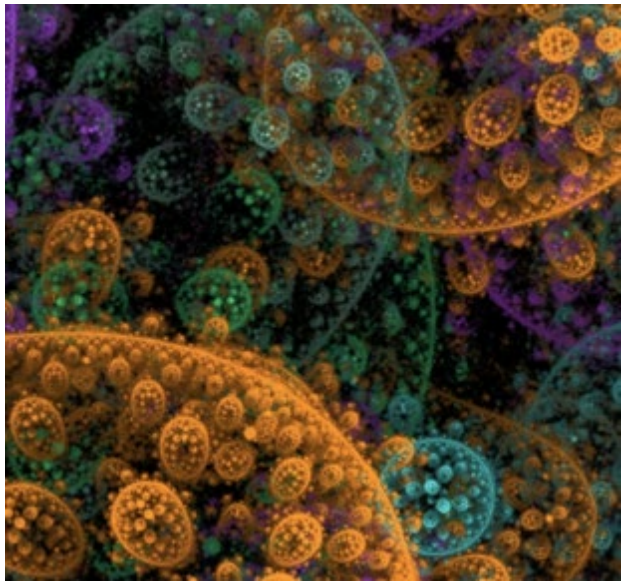
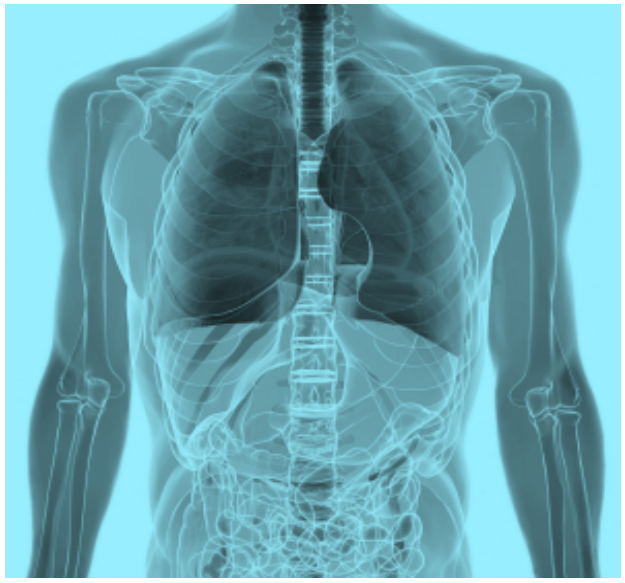
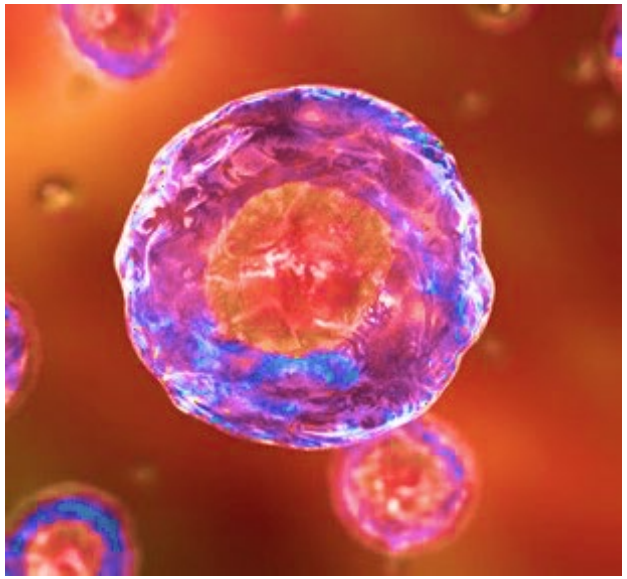
Galecto

Galecto Expert Perspectives: Myelofibrosis Treatment Landscape Current and Potential Future Treatments featuring Srdan Verstovsek, M.D., Ph.D.

June 9, 2021

Forward-looking statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding Galecto, Inc.'s (the "Company") strategy, future operations, future financial position, projected costs, prospects, plans, and objectives, are forward-looking statements. Such forward-looking statements include statements about the GALACTIC-1 trial, plans for continuing to enroll patients, working with investigators and regulatory authorities, the timing of completing enrollment and the initial unblinded data readout, GB0139's potential (including the effectiveness of the 3 mg dose), plans for clinical development and potential to market, as well as Galecto's product candidates and pipeline. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potential," "predict," "project," "target," "should," "would," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. For such statements, we claim the protection of the Private Securities Litigation Reform Act of 1995. The Company may not actually achieve the plans, intentions, or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements. Factors that could cause actual results to differ materially from such statements include, without limitation: our ability to modify the GALACTIC-1 trial protocol for GB0139 to the satisfaction of the FDA and other regulatory agencies; our ability to continue to enroll patients and complete the GALACTIC-1 trial with fewer dosage groups; the risk that FDA or other regulatory agencies impose a clinical hold on the GALACTIC-1 trial; that drug development is expensive, time consuming, uncertain and susceptible to change, interruption, delay or termination; the duration and severity of the ongoing coronavirus disease (COVID-19) pandemic, including but not limited to the impact on our clinical and other operations, the operations of our suppliers, others and the capital markets, which in each case remains uncertain; that the timing and outcome of research, development and regulatory review and feedback is uncertain; our need to raise additional capital to advance all of our programs; the amount of our future losses is uncertain and could cause our stock price to fluctuate or decline; topline data may not accurately reflect the complete results of a particle study or trial; results of clinical trials and other studies are subject to different interpretation and may not be predictive of future results; new data or results may be unexpected or unfavorable; our drug candidates may not advance in development or be approved for marketing; clinical trial and other studies may not proceed at the time or in the manner expected or at all; enrolling patients in our ongoing and intended clinical trials is competitive and challenging; clinical and nonclinical data is voluminous and detailed, and regulatory agencies may interpret or weigh the importance of data differently and reach different conclusions than we or others, request additional information, have additional recommendations or change their guidance or requirements; data and information related to our program may not meet regulatory requirements or otherwise be sufficient for further development at all or on our projected timeline; and other risks related to developing, seeking regulatory approval of and commercializing drugs, including regulatory, manufacturing, supply and marketing issues and drug availability. Additional factors that could cause results to differ materially from those stated or implied by our forward-looking statements are disclosed in our Securities and Exchange Commission (SEC) filings, including under the headings "Risk Factors." In addition, the forward-looking statements included in this presentation represent the Company's views as of the date of this presentation. The Company anticipates that subsequent events and developments will cause its views to change. However, while the Company may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so.

Investment Highlights

Publicly traded (NASDAQ: GLTO) biotech with differentiated focus on fibrosis & cancer

- Potentially first-in-class with FDA and EMA orphan drug designation (ODD) for lead asset GB0139
- Small-molecule fibrosis inhibitors targeting Galectin-3 & lysyl oxidase-like 2 (LOXL2)

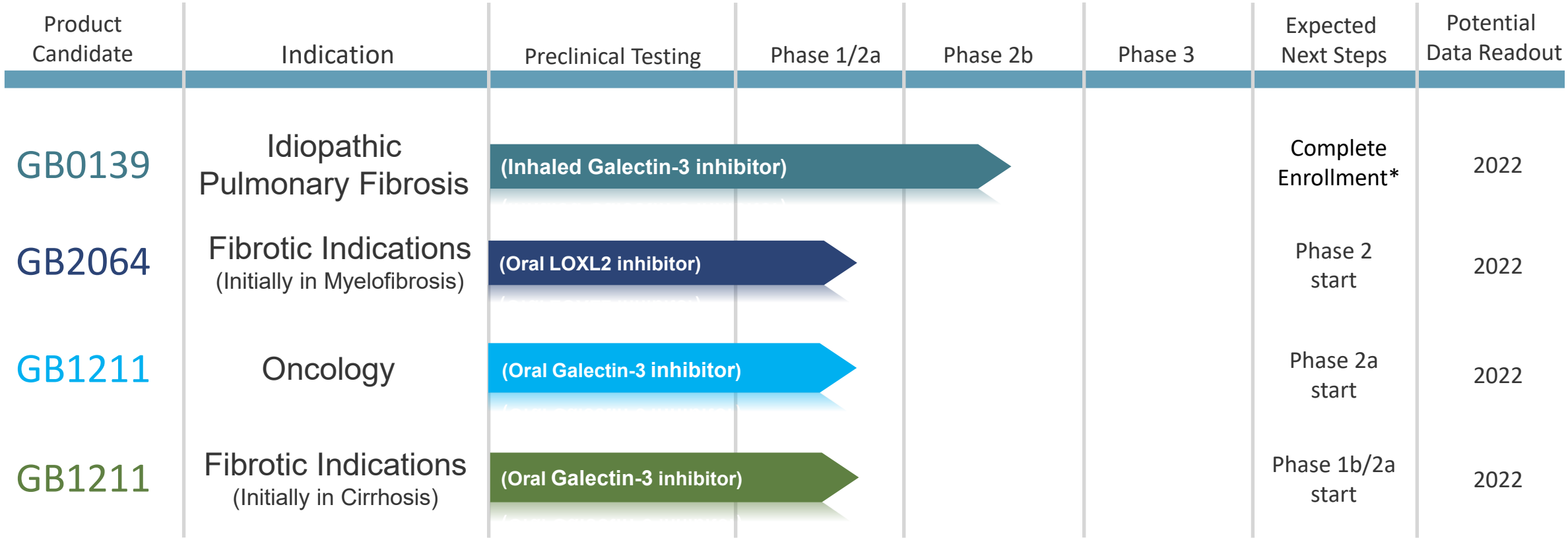
Strong pipeline with meaningful catalysts

- Phase 2b trial in idiopathic pulmonary fibrosis (IPF) ongoing
- Phase 2 studies in myelofibrosis, NSCLC and cirrhosis to be initiated later in 2021

Raised ~$160M in 2H 2020 – Cash balance (Mar 31, 2021) ~$149M, funded into 2024

- $95 million in October 2020 IPO led by BoA, SVB Leerink & Credit Suisse
- $64 million crossover round in September 2020

Unique Pipeline Targeting Fibrosis and Cancer



* protocol amendment in process

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center

Making Cancer History®

Current treatment landscape and unmet clinical needs for myelofibrosis

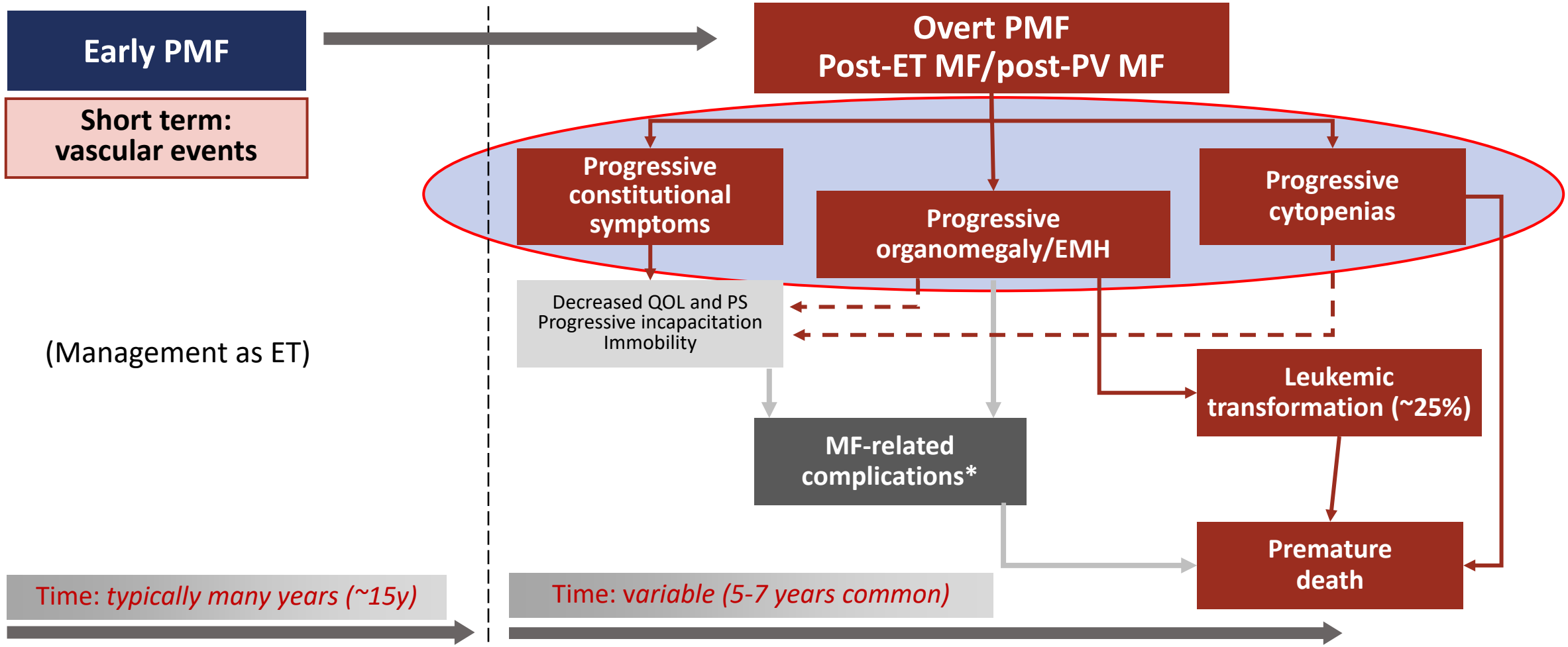
Srdan Verstovsek, M.D., Ph.D.

Professor of Medicine, Department of Leukemia

University of Texas, MD Anderson Cancer Center

Houston, Texas, USA

Myelofibrosis: Disease Course and Complications



Abbreviations: EMH, extramedullary hematopoiesis; ET, essential thrombocythemia; PMF, primary myelofibrosis; PS, performance status; PV, polycythemia vera; QOL, quality of life.

1. Mughal TI, et al. *Int J Gen Med.* 2014;7:89-101; 2. Haybar H, et al. *Cardiovasc Hematol Disord Drug Targets*. 2017;17(3):161-166.

MF is a Progressive Disease

After 1 year of diagnosis, significantly more patients have anemia, thrombocytopenia, circulating blasts, transfusion requirements, constitutional symptoms, splenomegaly, and unfavorable karyotype

	At Diagnosis (n = 340)	After 1 Year of Diagnosis (n = 386)
Hemoglobin < 10 g/dL	38%	64%
Platelet count < 100 × 10^9/L	18%	31%
Circulating blasts ≥ 1%	45%	66%
Requires transfusions	24%	45%
Constitutional symptoms	29%	34%
Splenomegaly > 10 cm	21%	46%
Unfavorable karyotype	10%	18%

Tefferi A, et al. *Mayo Clin Proc.* 2012;87:25-33.

Updated NCCN Guidelines for Treatment of MF: Based on Risk and Symptoms/Signs

Lower-risk

- Asymptomatic ➔ observation or clinical trial
- Symptomatic ➔ clinical trial or observation or **ruxolitinib** or **peginterferon α-2a** or **Hydroxyurea**

Higher-risk

- Transplant eligible ➔ allo-HSCT

or

- Transplant ineligible ➔ **ruxolitinib** or **fedratinib** or clinical trial

AND/OR

- Transplant ineligible/anemia ➔ anemia prescription or clinical trial

Lower-risk: MIPSS-70 ≤ 3; MIPPS-70+ ≤ 3; DIPSS-Plus ≤ 1; DIPSS ≤ 2; MYSEC-PM <14
Higher-risk: MIPSS-70 ≥ 4; MIPPS-70+ ≥ 4; DIPSS-Plus > 1; DIPSS > 2; MYSEC-PM ≥14

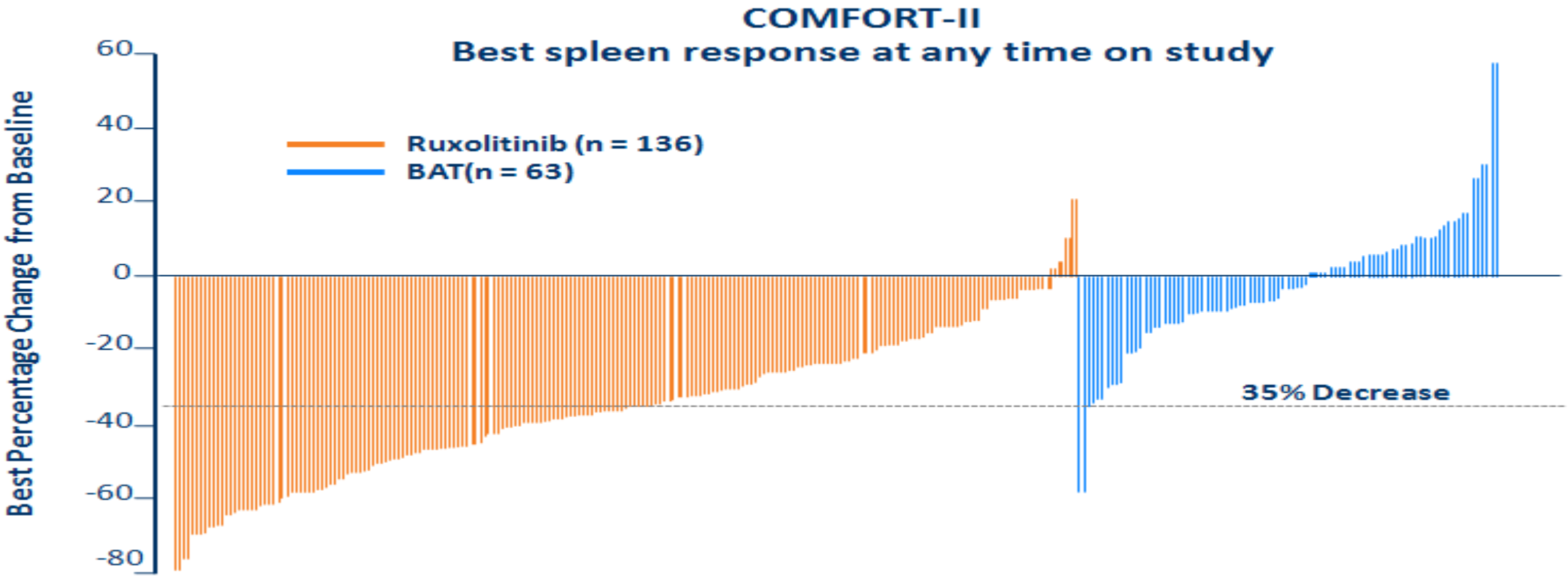
allo-HSCT, allogeneic hematopoietic stem cell transplantation; DIPSS, Dynamic International Prognostic Scoring System; Int, intermediate; MIPPS: Mutation-Enhanced International Prognostic Score System; MYSEC-PM, Myelofibrosis Secondary to PV and ET-Prognostic Model; NCCN, National Comprehensive Cancer Network.

National Comprehensive Cancer Network (NCCN). Myeloproliferative Neoplasms (Version 1.2020). Available from: www.nccn.org/professionals/physician_gls/pdf/mpn.pdf. Last accessed August 2020.

"Clinical needs" oriented current therapy for MF

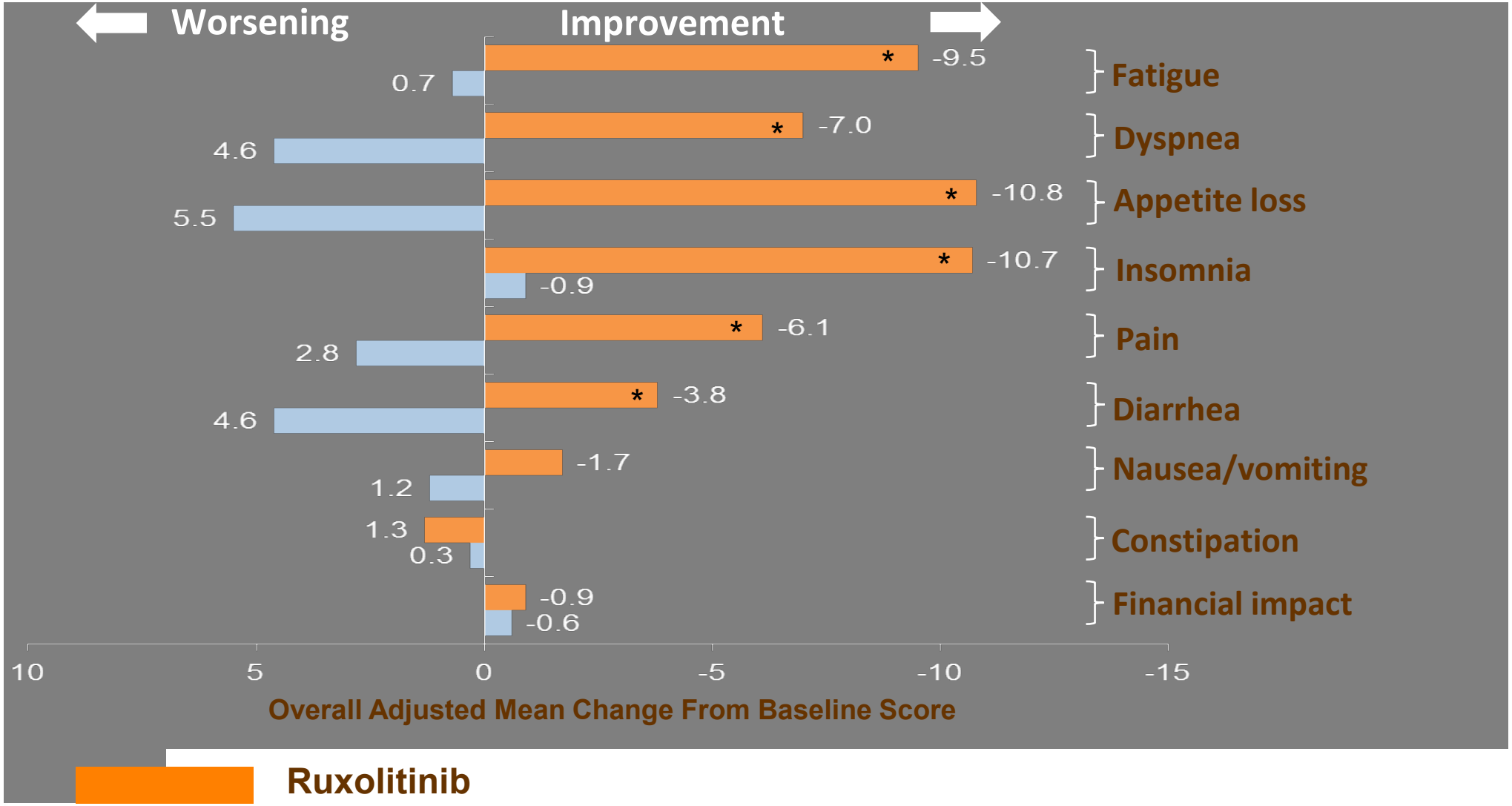
Clinical need	Drugs / Intervention	
Anemia	• Erythropoietin • Corticosteroids • Danazol	• Thalidomide • Lenalidomide
Symptomatic splenomegaly	• Ruxolitinib, Fedratinib, Hydroxyurea	• Cladribine, IMIDs • Splenectomy
Extramedulary hematopoiesis	• Radiation therapy	
Hyperproliferative (early) disease	• Interferon, hydroxyurea	
Risk of thrombosis	• Low-dose ASA	
Constitutional symptoms/ QoL	• Ruxolitinib, Fedratinib, Corticosteroids	
Accelerated/blastic Phase	• Hypomethylating agents	
Improved survival	• Allo SCT • Ruxolitinib	

Spleen Volume Response: Ruxolitinib vs. BAT

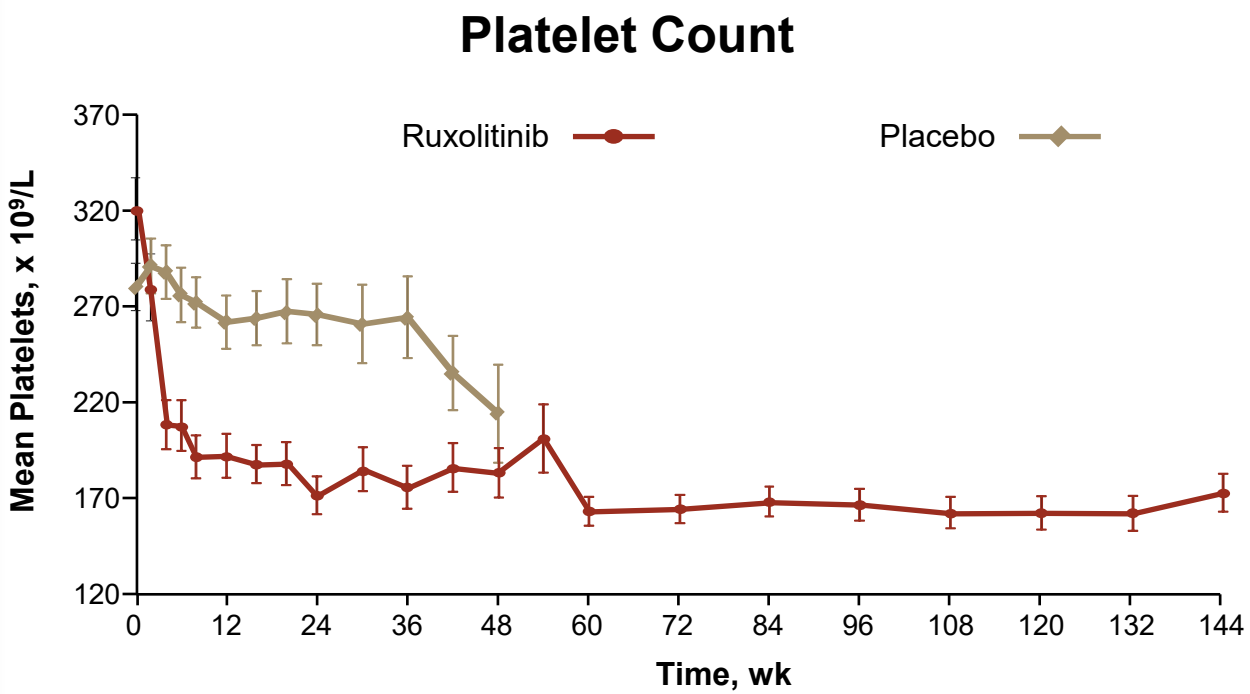


	Ruxolitinib	BAT
↓ Spleen volume	132 (97%)	35 (56%)
↑ Spleen volume	4 (3%)	28 (44%)

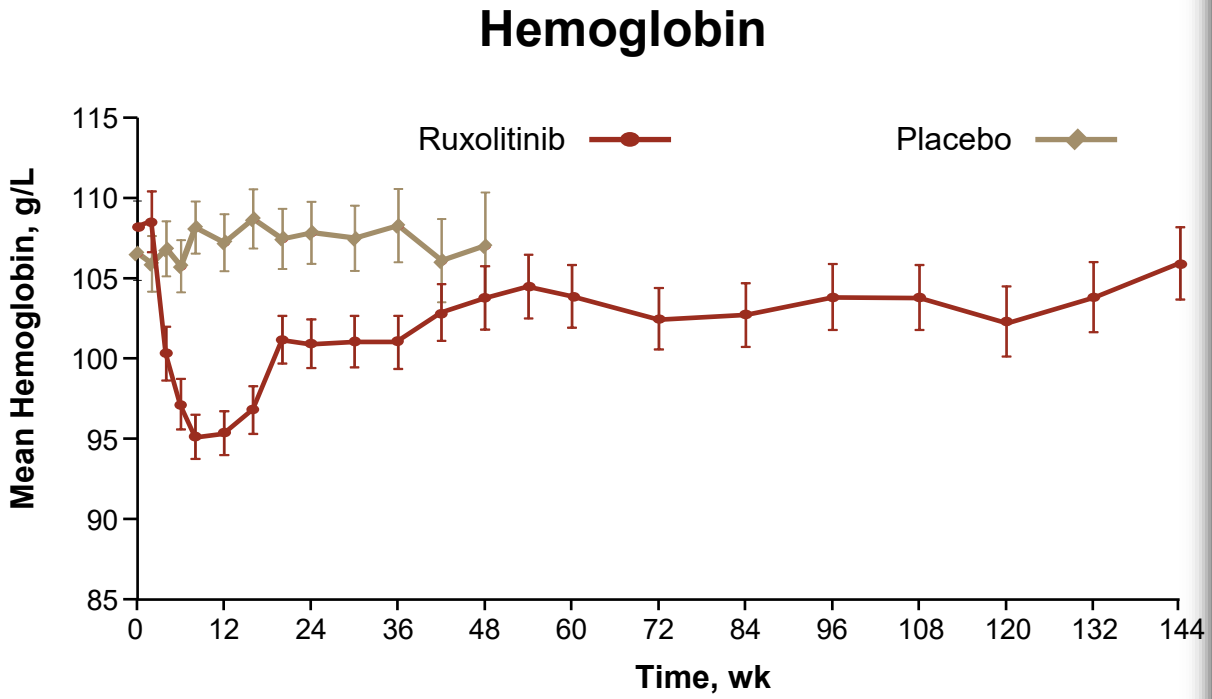
Improvement in Symptoms: Ruxolitinib vs. BAT



Mean Platelet Count and Hemoglobin Over Time COMFORT-I[1]



No. of Patients													
RUX	155	144	143	136	124	112	110	107	104	100	94	88	79
Placebo	151	128	112	82	37								

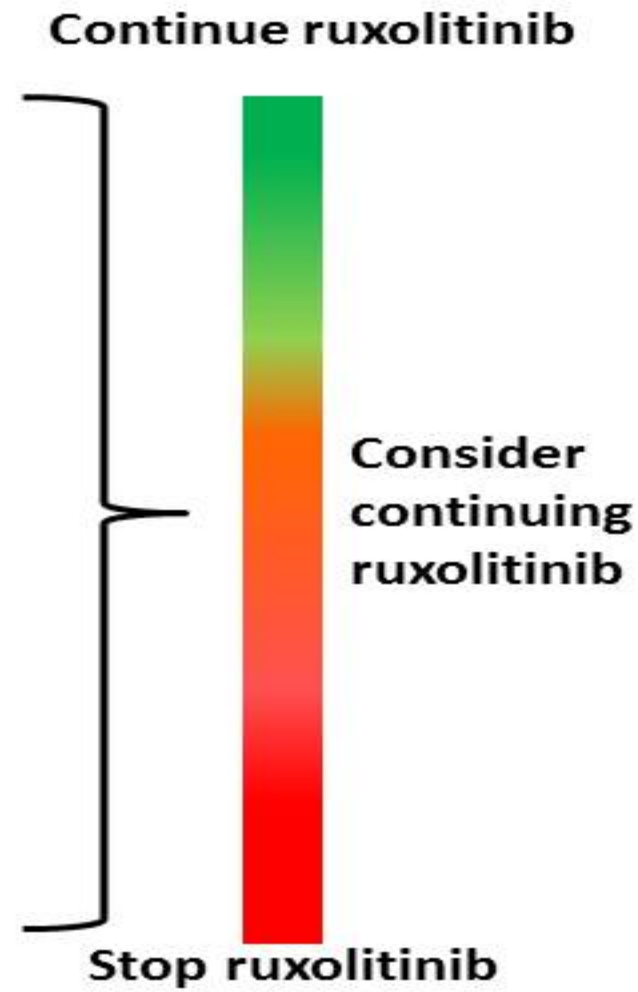


No. of Patients													
155	145	143	136	124	113	110	107	104	100	94	88	79
151	132	113	83	37								

1. Verstovsek S et al. *Haematologica*. 2015;100:479-488.

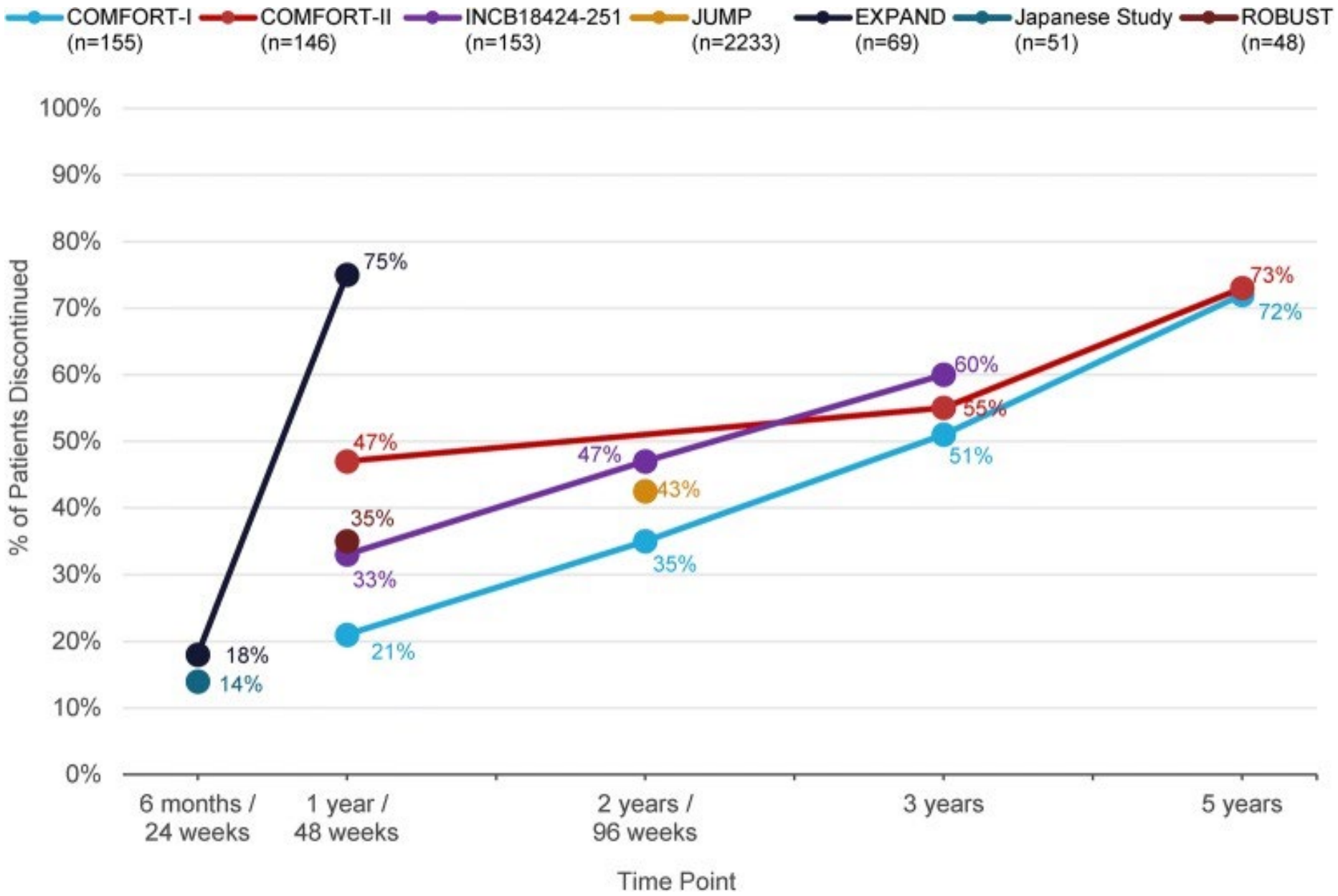
British Guidelines for myelofibrosis & use of JAK inhibitors

Symptoms	Spleen	Haematological toxicity
Clear	Clear	No
		Yes
	Suboptimal	No
		Yes
Suboptimal		No
		Yes
Minimal	Equivocal	No
		Yes
None	None	No
		Yes



Reilly JT, et al. British Journal of Haematology. 2014; 167 (3): 418-420

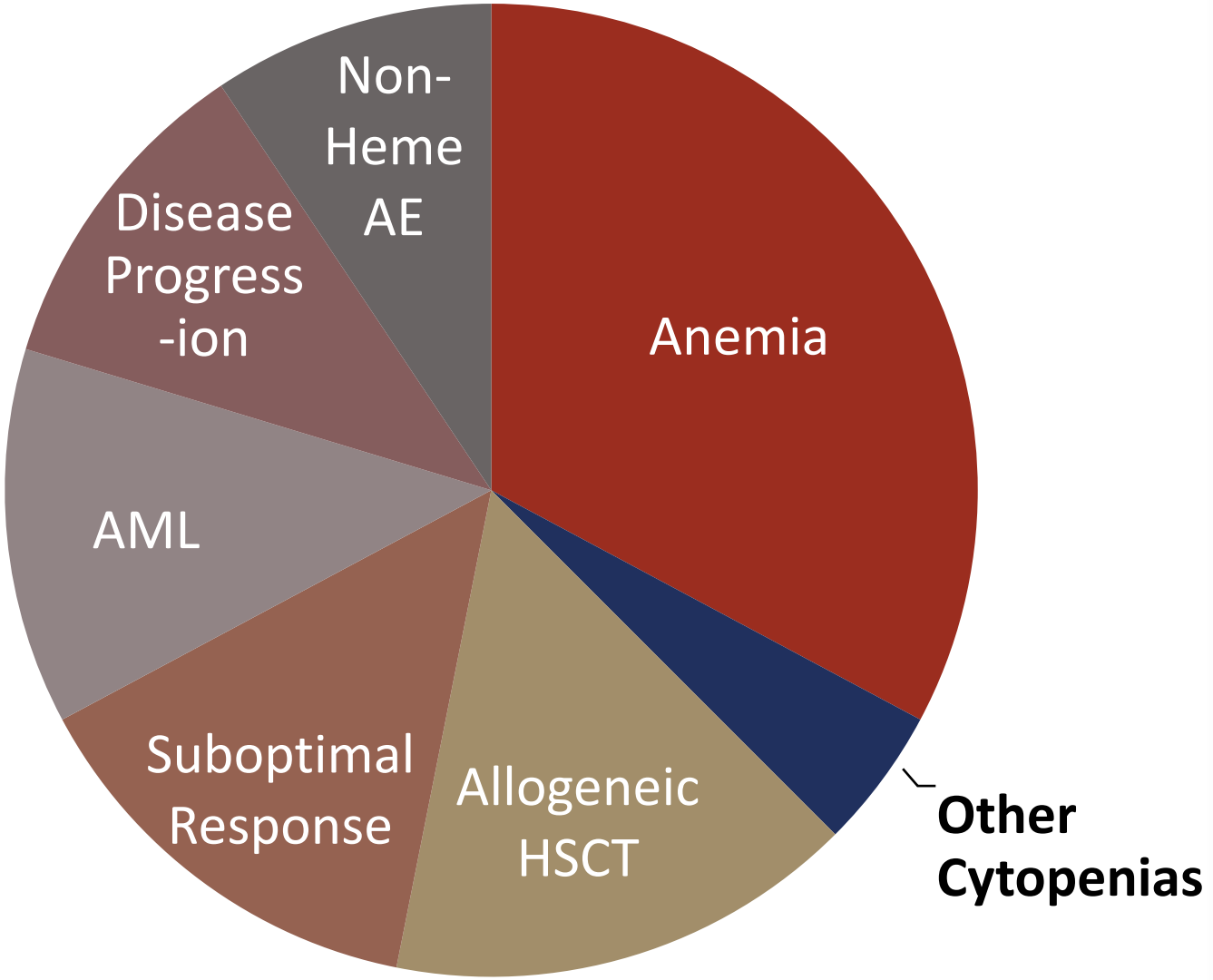
Duration of Ruxolitinib therapy



Harrison 2019

Reasons for stopping Ruxolitinib

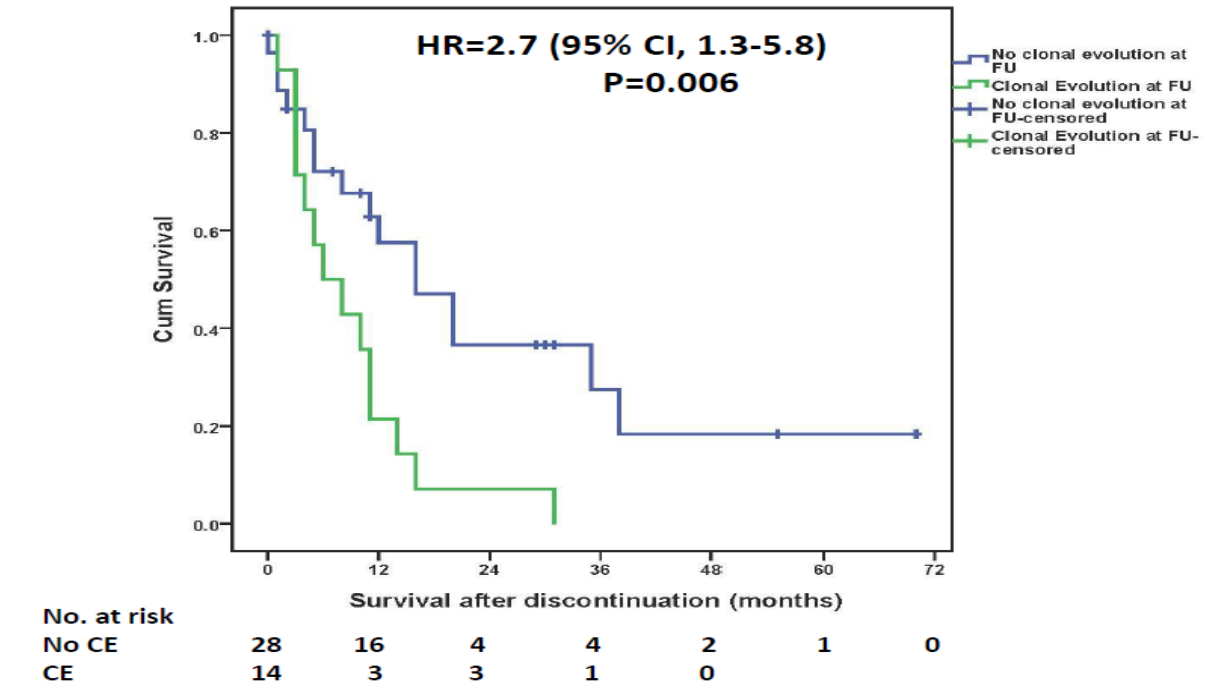
Anemia appears to be the leading cause of ruxolitinib discontinuations



Kuykendall AT, et al., *Annals of Hemotology* (2018) 97: 435-441.

Outcomes in MF after Ruxolitinib Discontinuation

- Survival after ruxolitinib d/c poor, median 14 months
- Shorter survival associated with low platelets
- 35% patients acquired a new mutation while receiving ruxolitinib (61% *ASXL1)*
- Patients showing clonal evolution had significantly shorter survival after d/c (6 vs 16 months, *P*=0.006)



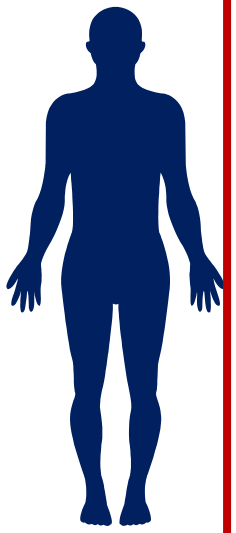
- Salvage therapy or re-challenge with ruxolitinib can provide responses after d/c.
- This continues to be an area of unmet clinical need in MF.[2]

1. Newberry KJ, et al. *Blood* 2017; 130(9):1125-1131; 3. Kuykendall AT, et al. *Ann Hematol.* 2018;97(3):435-441; 3. Gerds A, et al. *Clin Lymphoma Myeloma Leuk.* 2018;18(11):e463-e468.

Fedratinib in Myelofibrosis: JAKARTA and JAKARTA-2 Trials

Phase 3 JAKARTA Trial

Fedratinib vs placebo in patients with Int-2/high-risk MF first line

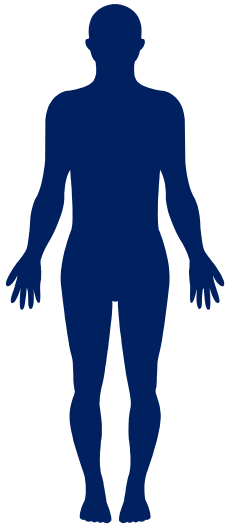


N = 289

Fedratinib 400 mg	Fedratinib 500 mg
37% Spleen volume reduction ≥ 35%	40% Spleen volume reduction ≥ 35%
40% Symptom burden reduction ≥ 50%	34% Symptom burden reduction ≥ 50%
14% Discontinuation due to AEs	25% Discontinuation due to AEs

Phase 2 JAKARTA-2 Trial

Fedratinib vs placebo in patients with Int-2/high-risk MF resistant or intolerant to ruxolitinib



N = 97
*N = 79

Primary analysis	Reanalysis (2019)*
55% Spleen volume reduction ≥ 35%	30% Spleen volume reduction ≥ 35%
	27% Symptom burden reduction ≥ 50%
11.0% Discontinuation due to AEs	

*More stringent criteria for relapse, refractory, and intolerance to ruxolitinib

- AE = Adverse Event; Int-2 = Intermediate-2
- Pardanani A, et al. *JAMA Oncol*. 2015;1(5):643–651; Harrison CN, et al. *Lancet Haematol*. 2017;4(7):317–324; Harrison CN, et al. ASCO 2019. Abstract 7057.

Fedratinib Adverse Events: JAKARTA Trial

Adverse Event, %	Fedratinib 400 mg (n = 96)		Fedratinib 500 mg (n = 97)		Placebo (n = 95)	
	All Grades	Grade 3 or 4	All Grades	Grade 3 or 4	All Grades	Grade 3 or 4
Nonhematologic						
Diarrhea	66	5	56	5	16	0
Vomiting	42	3	55	9	5	0
Nausea	64	0	51	6	15	0
Constipation	10	2	18	0	7	0
Asthenia	9	2	16	4	6	1
Abdominal pain	15	0	12	1	16	1
Fatigue	16	6	10	5	1	0
Hematologic						
Anemia	99	43	98	60	91	25
Thrombocytopenia	63	17	57	27	51	9
Lymphopenia	57	21	66	27	54	21
Leukopenia	47	6	53	16	19	3
Neutropenia	28	8	44	18	15	4

Black box warning

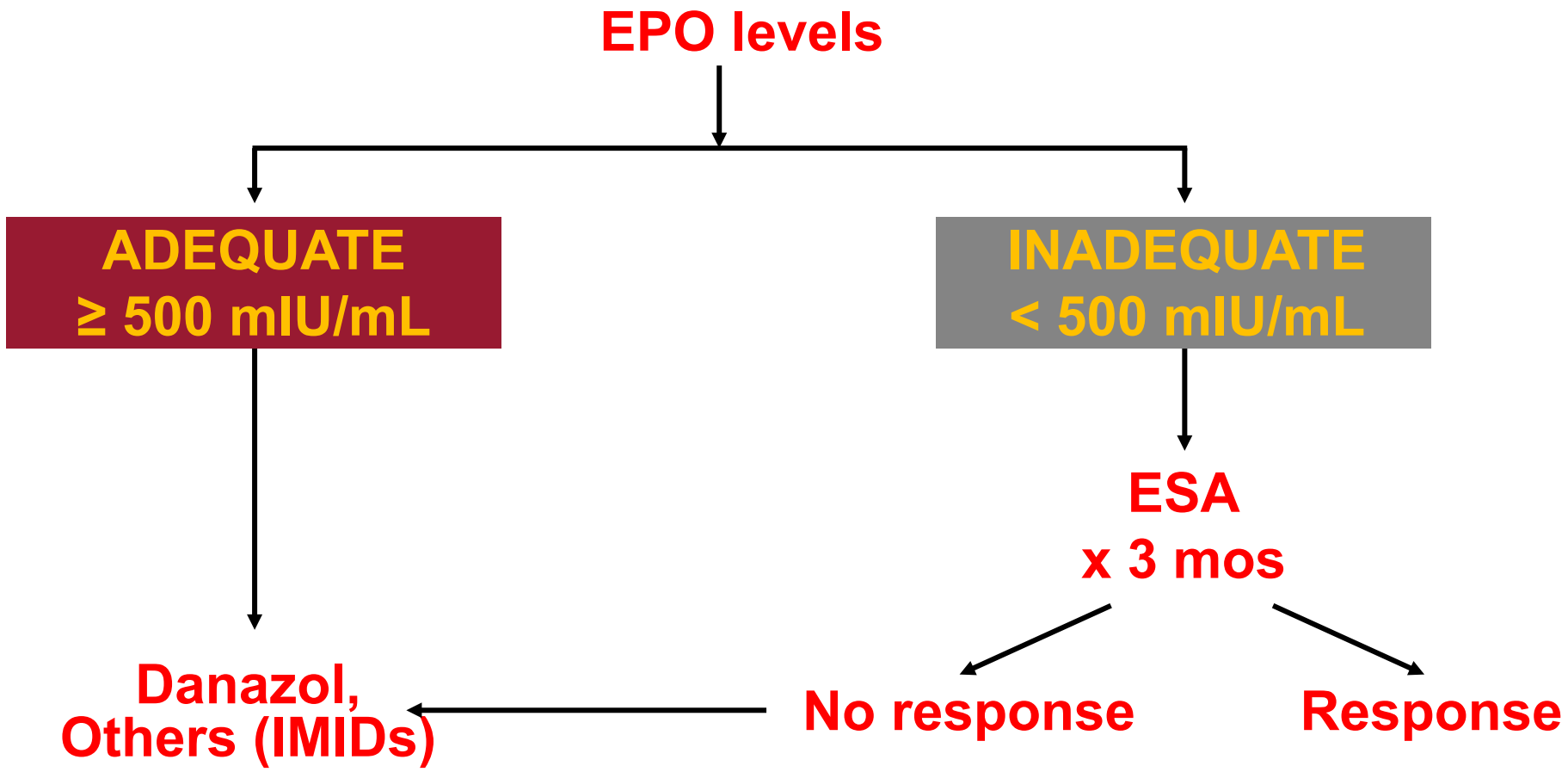
- Wernicke's encephalopathy (ataxia, altered mental status, ophthalmoplegia) occurred in 8 of 608 (1.3%) patients receiving fedratinib in clinical trials

Considerations

- Measure and address thiamine levels prior to treatment initiation
- Do not start fedratinib in patients with thiamine deficiency

Pardanani A, et al. *JAMA Oncol*. 2015;1(5):643–651.

Approach to the Treatment of Anemia in MF



Approach to the Treatment of Thrombocytopenia in MF

Splenectomy in MF

ASSOCIATED RISKS

- 40% morbidity
- 10% mortality
- Liver enlargement and failure
- Higher acute transformation rate?
- Average survival post splenectomy: 18 months

CONTRAINDICATION

Thrombocytosis

MAIN INDICATIONS

- Symptomatic splenomegaly unresponsive to treatment
- Severe refractory anemia and thrombocytopenia
- Unresponsive constitutional symptoms
- Uncontrollable hemolysis
- Portal hypertension

Selected JAKi-Based Rational Combinations

	Drug	Mechanism of Action	Phase
Accelerated/blastic phase	Azacitidine	HMA	2
	Decitabine	HMA	2
Cytopenia (ANEMIA)	Luspatercept	Activin receptor ligand rap	3
	Danazol	Androgen	2
	Thalidomide	IMiD	2
	Pomalidomide	IMiD	1/2
Higher Responses In Spleen and Symptoms	PEG-IFNa-2a	–	1/2
	PU-H71	HSP90i	1/2
	Itacitinib	JAK1i	2
	Navitoclax	BCL-2/BCL-xL	3
	Parsaclisib	PI3Kδi	3
	KRT-232	MDM2i	3
	CPI-0610	BETi	3
	Pevonedistat	NAEi	1

Selected Novel Single Agent Trials in Myelofibrosis

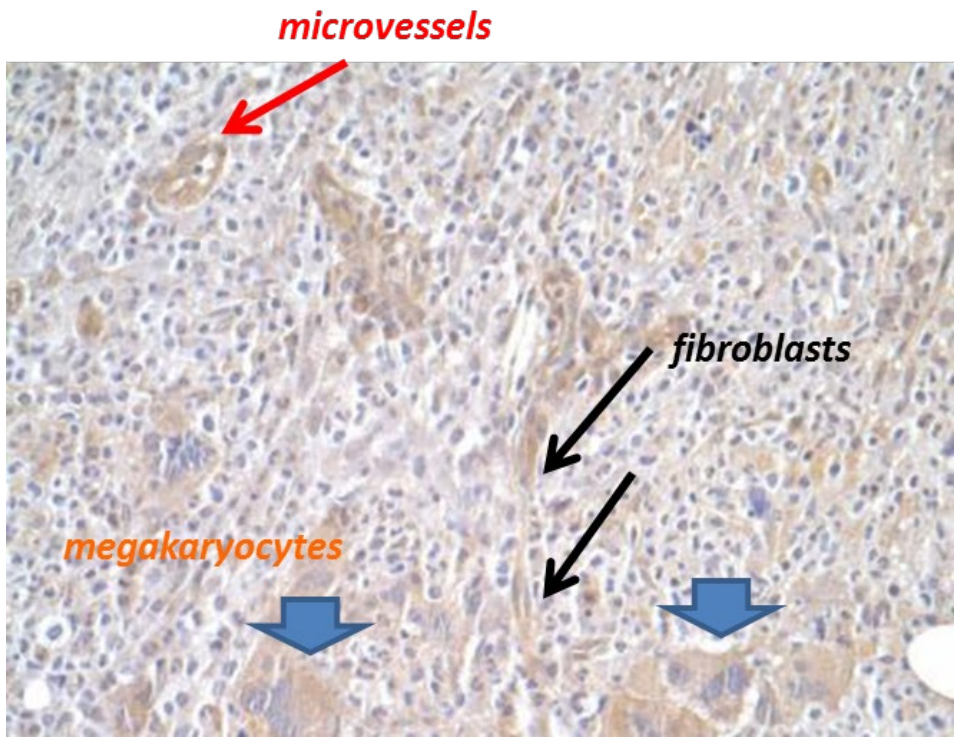
Agent	Drug Class	Phase
LCL-161	Smac-mimetic (IAP antagonist)	2
Momelotinib and pacritinib	JAK inhibitors	3
Tagraxofusp	CD123-targeting fusion protein	2
PRT543	PRMT5 inhibitor	1/2
Bomedemstat	LSD1 inhibitor	1/2
INCB054828	ALK inhibitor	1/2
Imetelstat	Telomerase inhibitor	3
PRM-151	Recombinant human pentraxin-2	2
Nivolumab/pembrolizumab	Anti-PD1 antibodies	2
Selinexor	SINE	2

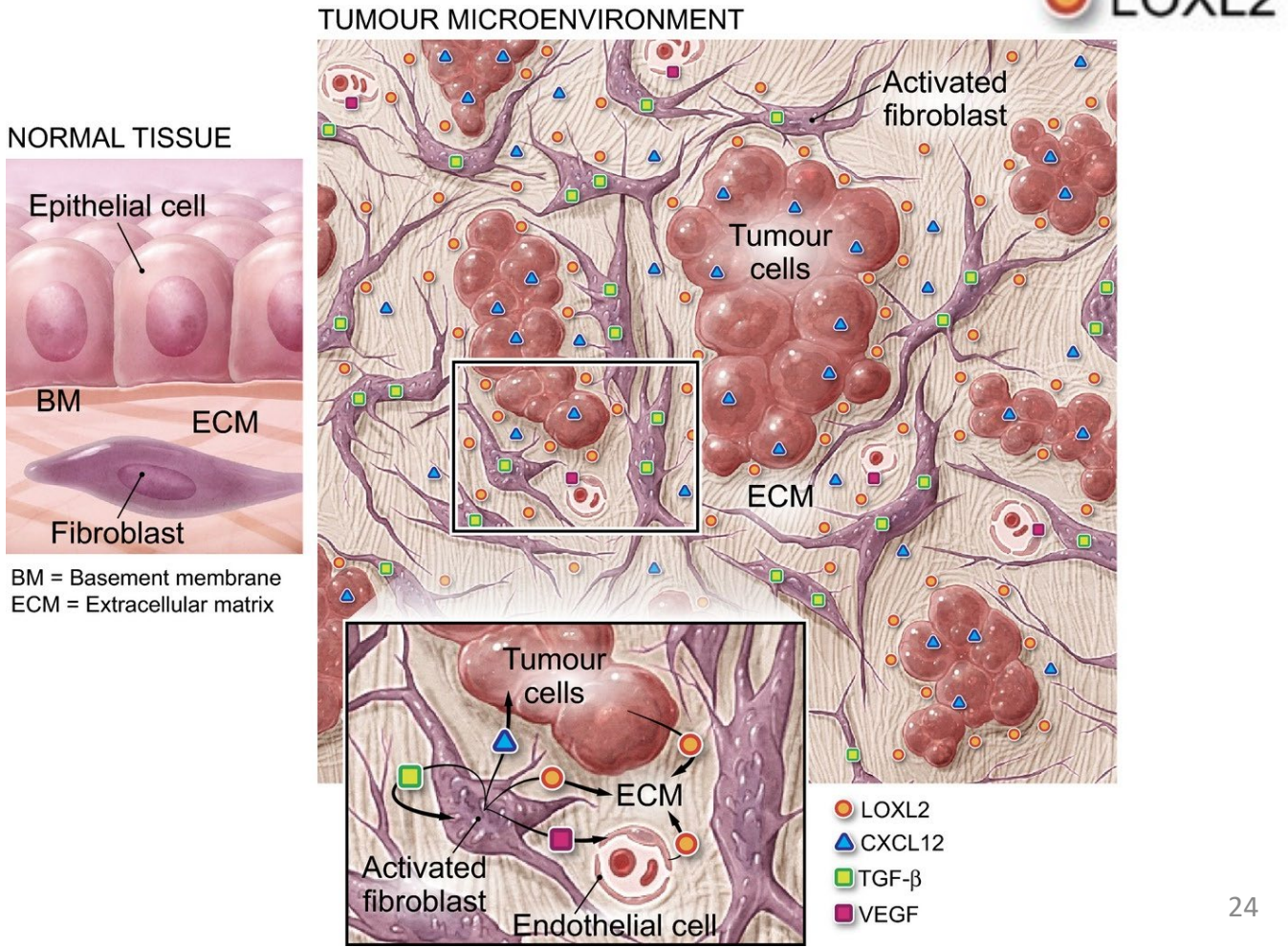
Bose P, Verstovsek S. *Leuk Lymphoma.* 2020;61(8):1797-1809.

LOXL2 in myelofibrosis

- LOXL2 is an enzyme that catabolizes the formation of lysine cross-linking in fibrillar collagens and elastins in the extracellular matrix
- LOXL2 is upregulated in Myelofibrosis and drives formation of BM fibrosis

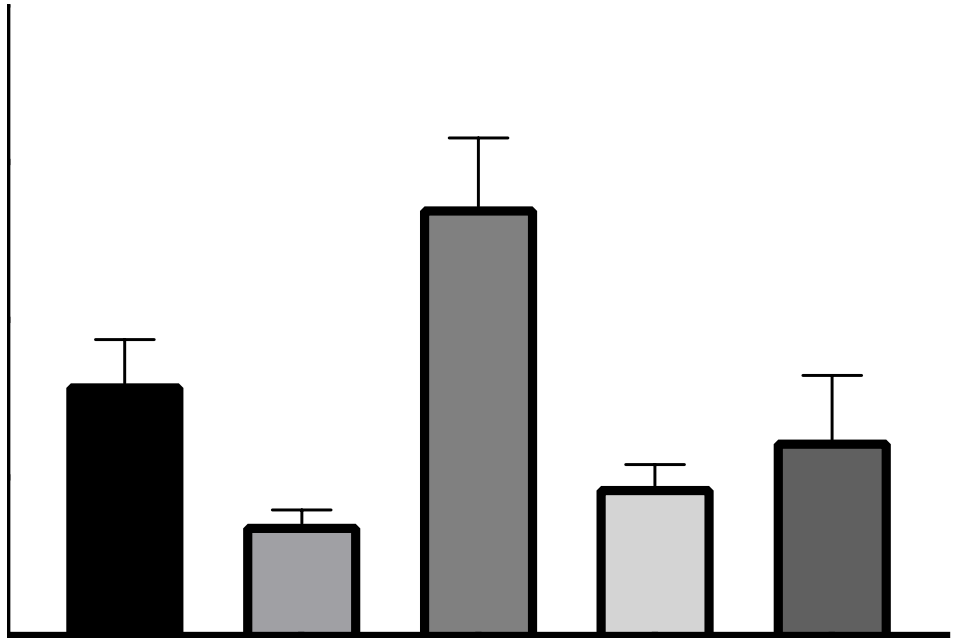
LOXL2 Staining in MF-BM



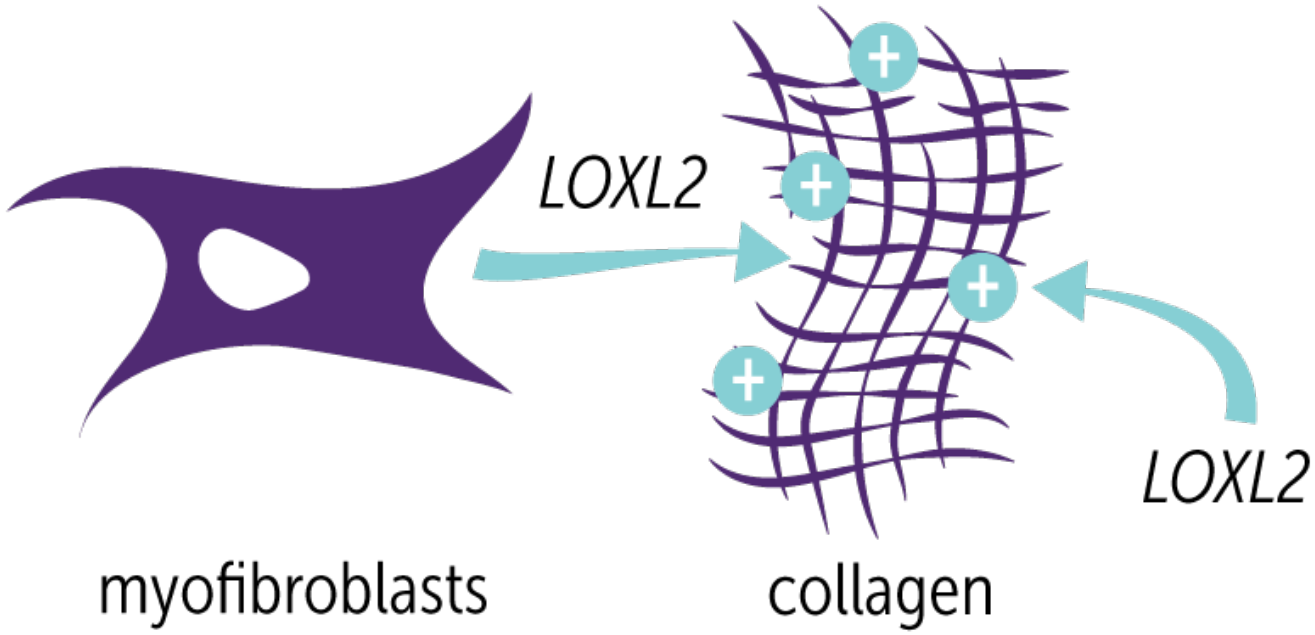


British Journal of Haematology, Volume: 176, Issue: 6, Pages: 939-949, 2017,

Targeting fibrosis via the LOXL2 mechanism



LOX Family Gene Expression in Myelofibrosis Stromal Cells



GB2064 in myelofibrosis bone marrow (BM)

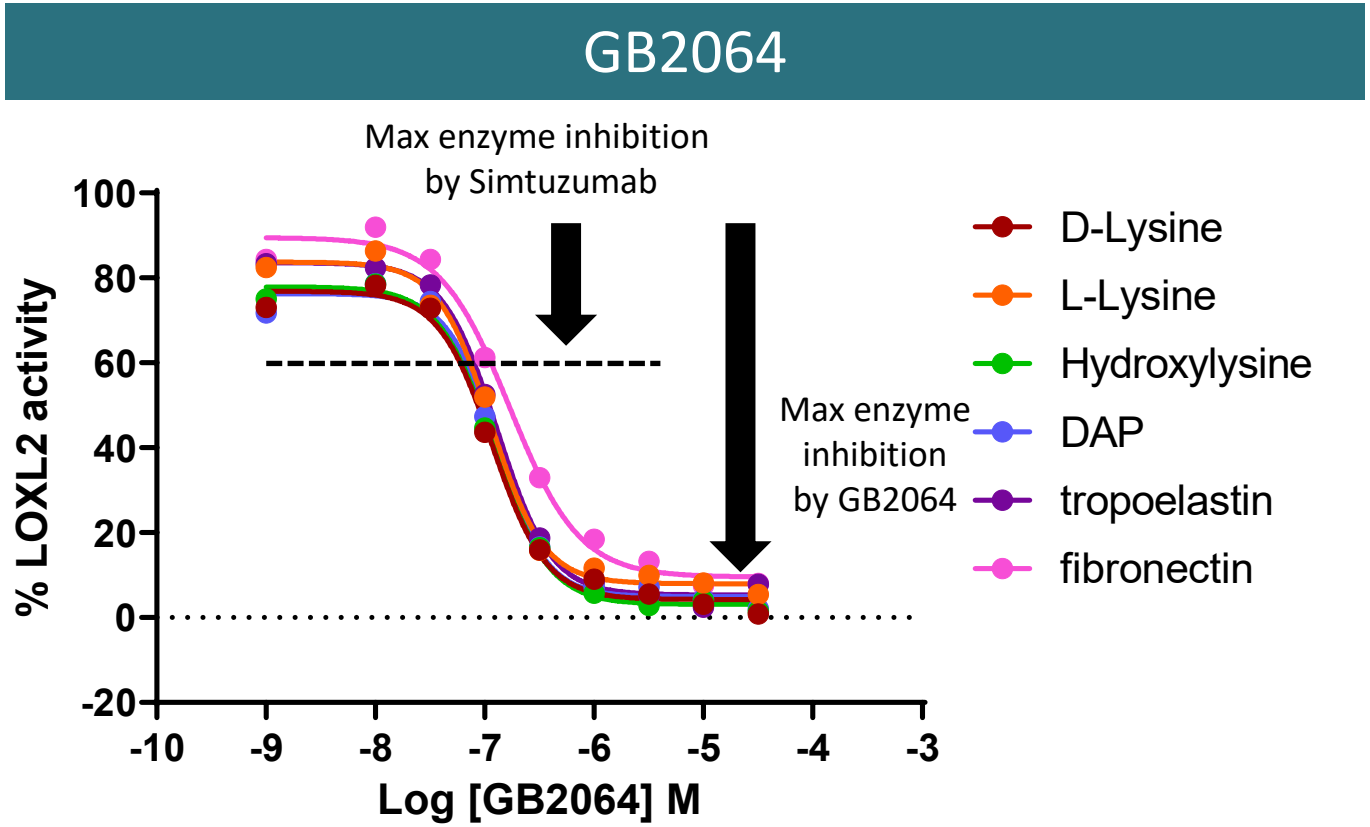
- The pseudoirreversible inhibition of the LOXL2 enzyme by GB2064 during **C-max,** and its fast clearance (T½ cirka 3 hours) enables
  - A hit and run enzyme inhibition
  - long duration effect in the bone marrow independent of the plasma concentration
  - Low systemic toxicity, due to low systemic drug levels for most of the time
  - Different than Simtuzumab (LOXL2 antibody previously tested in a clinical study)

- The potential effect of GB2064 in the BM may lead to
  - Reduced fibrotic bulk and more blood formation space
  - LOXL2 inhibition may have direct anti-cancer effects via reduced H3K4 oxidation (1) and via decreased stiffness of the extracellular matrix (2)
  - Increased blood formation and slower progression of the disease, i.e. be disease modifying

1) Cebrià-Costa, J.P., Pascual-Reguant, L., Gonzalez-Perez, A. *et al.* LOXL2-mediated H3K4 oxidation reduces chromatin accessibility in triple-negative breast cancer cells. *Oncogene* **39,** 79–121 (2020)
2) Dinca SC, Greiner D, Weidenfeld K, Bond L, Barkan D, Jorcyk CL. Novel mechanism for OSM-promoted extracellular matrix remodeling in breast cancer: LOXL2 upregulation and subsequent ECM alignment. Breast Cancer Res. 2021 May 19;23(1):56

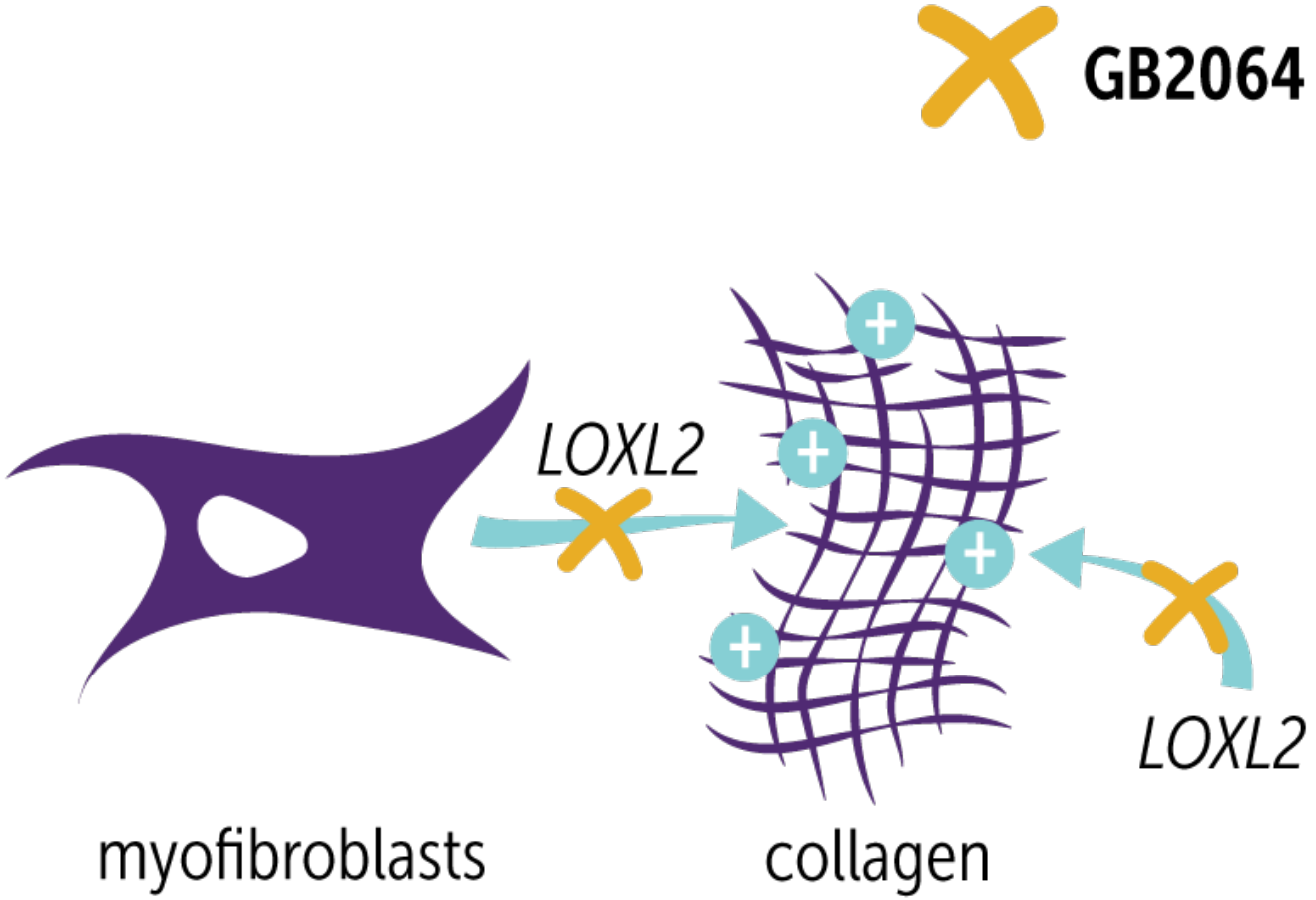
GB2064: Demonstrated *in vitro* inhibition of LOXL2

- GB2064 is a small-molecule inhibitor of the LOXL2 enzyme catalytic site, not an antibody approach
- GB2064 therefore has the potential to avoid the *in vivo* target low tissue penetration and low target engagement seen with Gilead's simtuzumab
- Simtuzumab maximal enzyme inhibition of 40% at the clinically unattainable 1µM level
- GB2064's superior efficacy to simtuzumab has been observed in cell-based assays and preclinical models

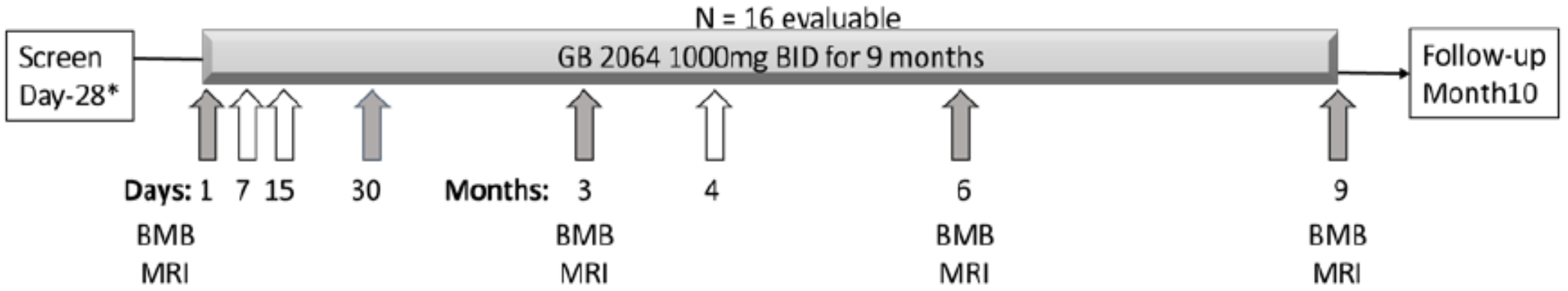


GB2064 fully inhibits LOXL2 activity using a variety of substrates

Targeting fibrosis via the LOXL2 mechanism



The MYLOX-1 study design and read-outs



- 16 evaluable patients (ineligible for or previously Rx with a JAKi)
- Open label GB2064
- 9 month therapy with GB2064 1g BID
- Biopsy at visit every 3 months
- additional safety and tolerability assessments performed at D7, D15, and M4

- Interim read after 8 evaluable patients completed 6 months of therapy and BMB.

- Bone Marrow biopsy (BMB) and MRI scan at Day 1, and then every 3 months
- Spleen volume
- BM fibrosis (central read)
- GB2064 assessment in BM tissue
- Clinical variables
  - Safety and Tolerability
  - Clinical activity assessments
  - PK/ PD
  - Biomarker assessments
  - Transfusion dependency
  - DIPSS plus assessment
  - MPN 10 and QoL assessments

GB2064: Phase 2a in myelofibrosis - Summary

- Ample evidence for central role of increased LOXL2 activity in myelofibrosis
- GB2064 potently inhibits LOXL2 in a pseudoirreversible manner, and shows antifibrotic activity in numerous models

- Upcoming Phase 2a trial is designed to generate both target engagement and efficacy data
- Opportunity for both orphan drug designation and fast track designation

- IND approved October 2020, and Phase 2a trial is about to start
  - Phase 1 SAD/MAD study completed in healthy volunteers
    - Safe and well tolerated
    - Reproducible PK suitable for twice daily dosing
  - Chronic toxicology studies completed

Thank You

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GB2064: Oral LOXL2 Inhibitor in Myelofibrosis

Overview and Treatment Opportunity

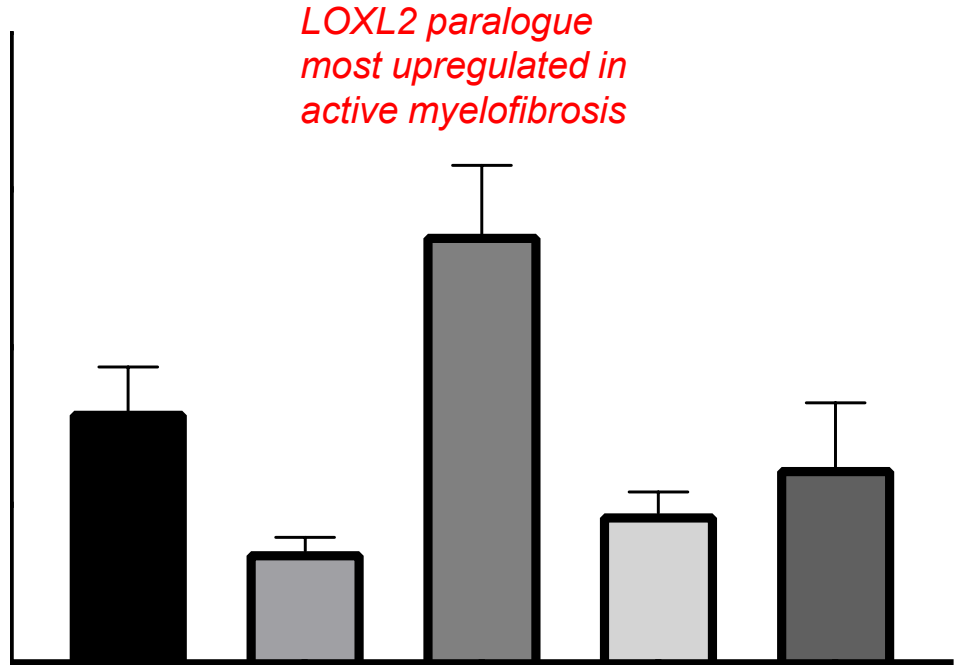
GB2064 (previously PAT-1251)

- A small-molecule inhibiting LOXL2, an enzyme that catabolizes the formation of lysine cross-linking in fibrillar collagens
- Potentially disease modifying
- Opportunity in multiple fibrotic indications

Myelofibrosis:

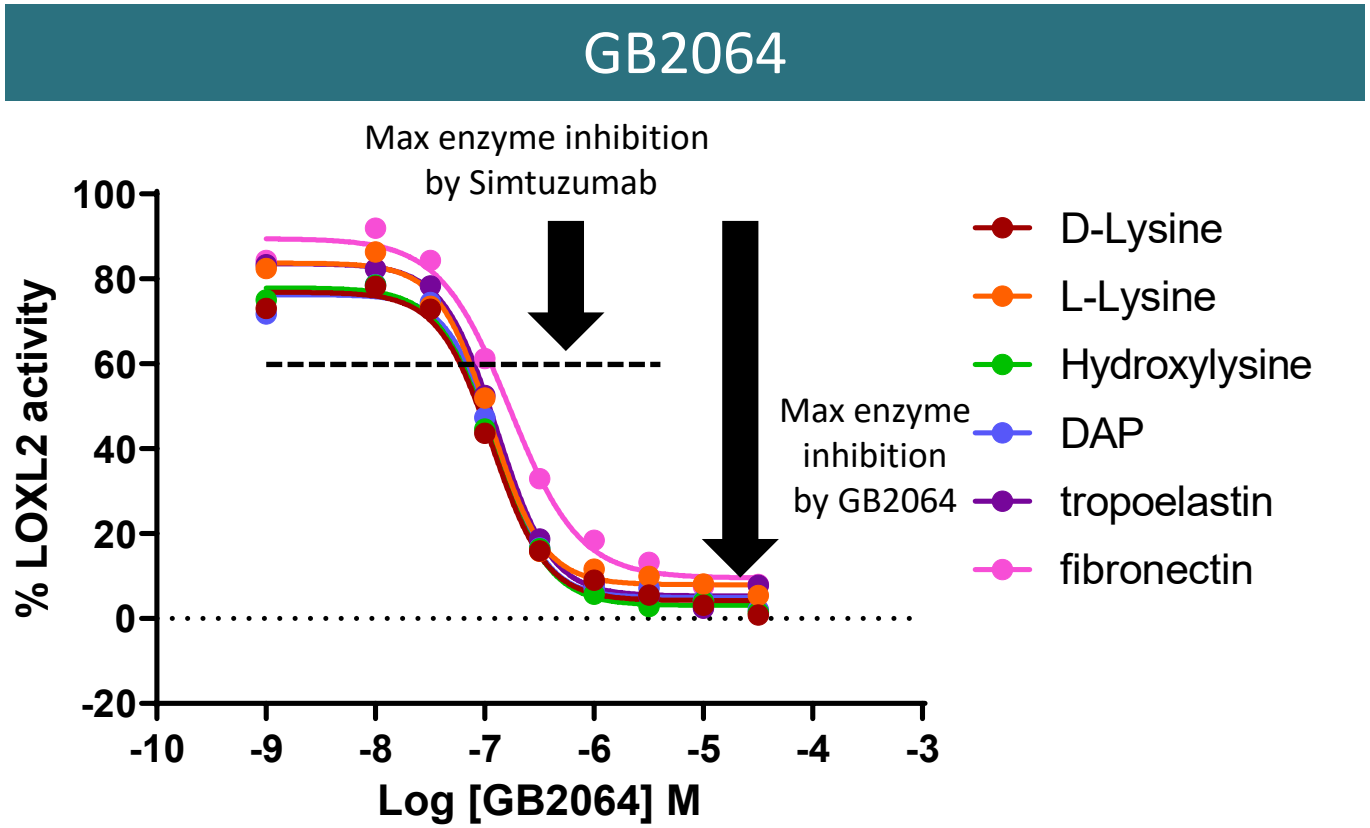
- Orphan indication: 16,000 - 18,500 patients in US
- Current therapies (JAK inhibitors) are not disease modifying
- Large market - Incyte's Jakafi achieved sales of $1.9B & $1.7B in 2020 & 2019, respectively

LOX Family Gene Expression in Myelofibrosis Stromal Cells



GB2064: Demonstrated *In Vitro* Inhibition of LOXL2

- GB2064 is a small-molecule inhibitor of the LOXL2 enzyme catalytic site, not an antibody approach
- GB2064 therefore has the potential to avoid the *in vivo* target low tissue penetration and low target engagement seen with Gilead's simtuzumab
- Simtuzumab maximal enzyme inhibition of 40% at the clinically unattainable 1µM level
- GB2064's superior efficacy to simtuzumab has been observed in cell-based assays and preclinical models



GB2064 fully inhibits LOXL2 activity using a variety of substrates

MYLOX-1: GB2064 in Myelofibrosis

- First patient dosing expected in coming months
- Single arm, open label study allowing real-time read of safety and activity
  - Planned for 16 evaluable patients initially for 9 months of treatment
- Patients who are ineligible for JAK-inhibitors or who do not tolerate JAKi
- Planned endpoints for readout include:
  - Blood formation
  - Bone marrow general histology and fibrosis
  - Demonstration of drug levels in target tissue
  - Imaging for spleen and liver volume

GB2064: Phase 2a in Myelofibrosis Summary

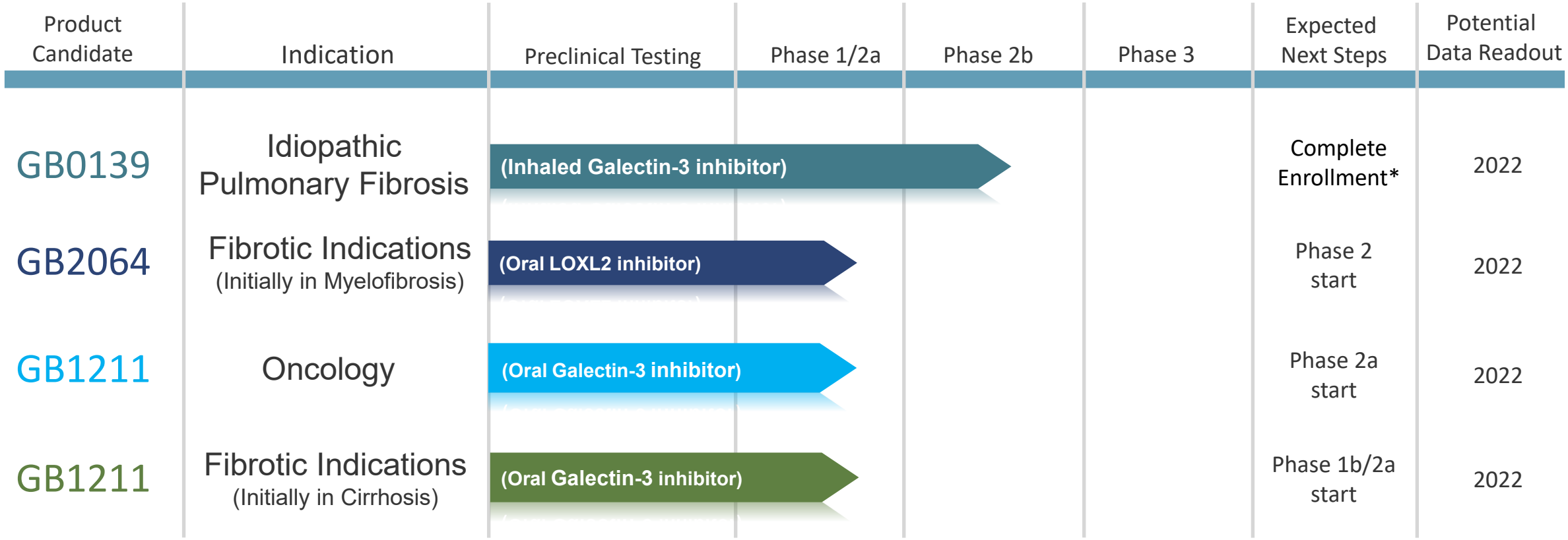
- Ample evidence for central role of LOXL2 in fibrosis
- GB2064 potently inhibits LOXL2 and shows antifibrotic activity in numerous models

- Upcoming Phase 2a trial could generate both target engagement and efficacy data in the same study as repeated biopsies are already standard practice
  - Opportunity for both orphan drug designation and fast track designation following data in this indication

- IND approved October 2020, and Phase 2a trial to start in coming months
  - Phase 1 SAD/MAD study already completed
  - Chronic toxicology studies completed
  - Robust efficacy in lung, liver and kidney models

Q&A

Unique Pipeline Targeting Fibrosis and Cancer



* protocol amendment in process