THE UNIVERSITY OF TEXAS
MD ANDERSON
CANCER CENTER

Managing Myelofibrosis (MF) in 2025

14th Joyce Niblack Memorial Conference on MPNs
Feb 15-16, 2025 | MPN Education Foundation
Phoenix, AZ

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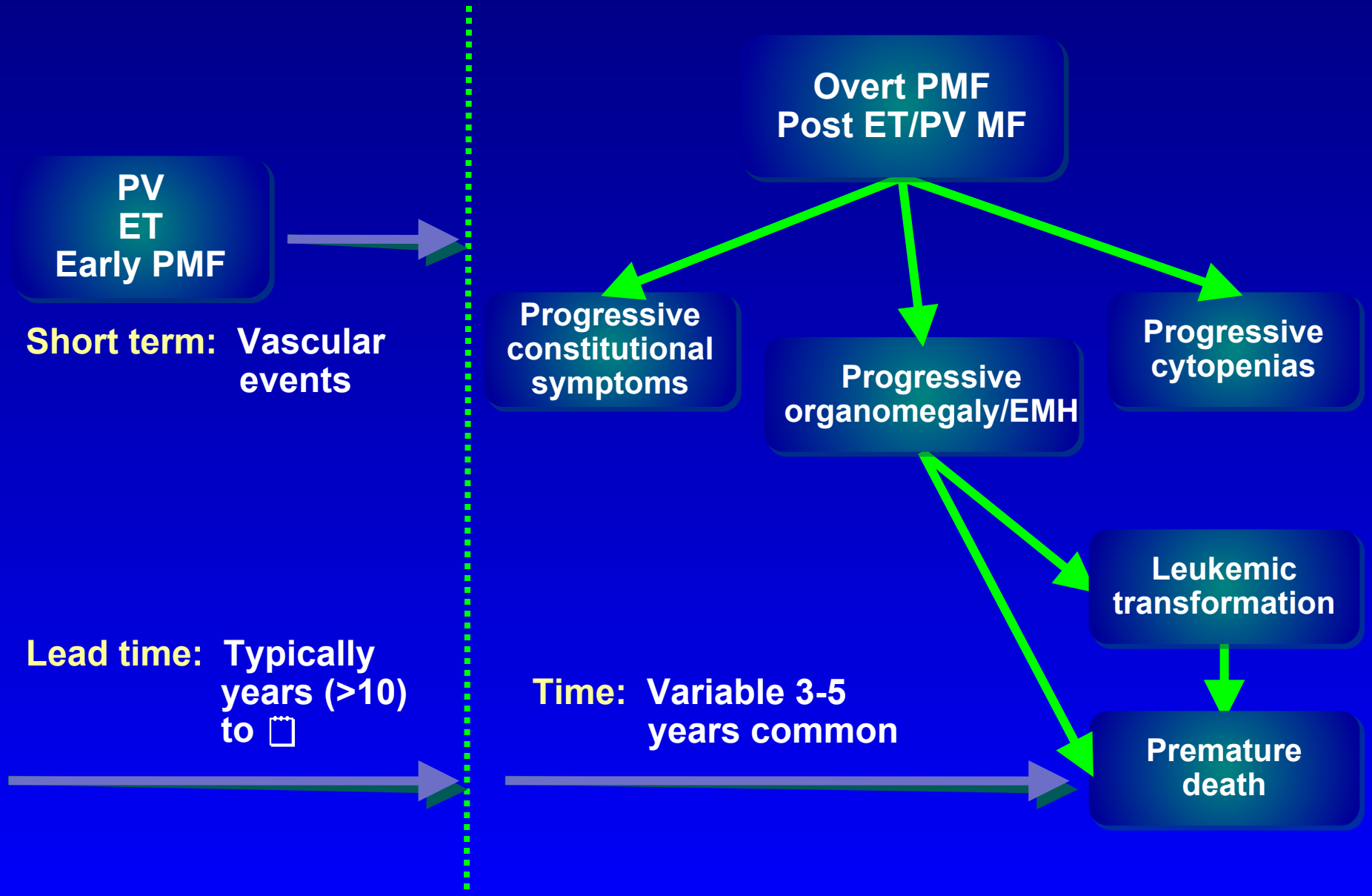
Natural History of (MPDs) MPNs

courtesy Dr Ruben Mesa, MD



JAK2 V617F Mutation

March 18th, 2005

Acquired mutation of the tyrosine kinase JAK2 in human myeloproliferative disorders

E Joanna Baxter*, Linda M Scott*, Peter J Campbell*, Clare East, Nasios Fourouclas, Sohelia Swanton, George S Vassiliou, Anthony J Bench, Elaine M Boyd, Natosha Curtin, Mike A Scott, Wendy N Erber, the Cancer Genome Project†, Anthony R Green

March 24th, 2005

Activating mutation in the tyrosine kinase JAK2 in polycythemia vera, essential thrombocythemia, and myeloid metaplasia with myelofibrosis

Ross L. Levine,[1,2,11] Martha Wadleigh,[2,11] Jan Cools,[6] Benjamin L. Ebert,[2,8] Gerlinde Wernig,[1] Brian J.P. Huntly,[1] Titus J. Boggon,[4] Iwona Wlodarska,[6] Jennifer J. Clark,[1] Sandra Moore,[1] Jennifer Adelsperger,[1] Sumin Koo,[1] Jeffrey C. Lee,[8] Stacey Gabriel,[8] Thomas Mercher,[1] Alan D'Andrea,[3] Stefan Fröhling,[1] Konstanze Döhner,[7] Peter Marynen,[6] Peter Vandenberghe,[6] Ruben A. Mesa,[9] Ayalew Tefferi,[9] James D. Griffin,[2] Michael J. Eck,[4] William R. Sellers,[2,8] Matthew Meyerson,[2,8] Todd R. Golub,[5,8,10] Stephanie J. Lee,[2,*] and D. Gary Gilliland[1,2,10,*]

March 27th, 2005

A unique clonal *JAK2* mutation leading to constitutive signalling causes polycythaemia vera

Chloé James[1*], Valérie Ugo[1,2,3*], Jean-Pierre Le Couédic[1*], Judith Staerk[4], François Delhommeau[1,3], Catherine Lacout[1], Loïc Garçon[1], Hana Raslova[1], Roland Berger[5], Annelise Bennaceur-Griscelli[1,6], Jean Luc Villeval[1], Stefan N. Constantinescu[4], Nicole Casadevall[1,3] & William Vainchenker[1,7]

April 28th, 2005

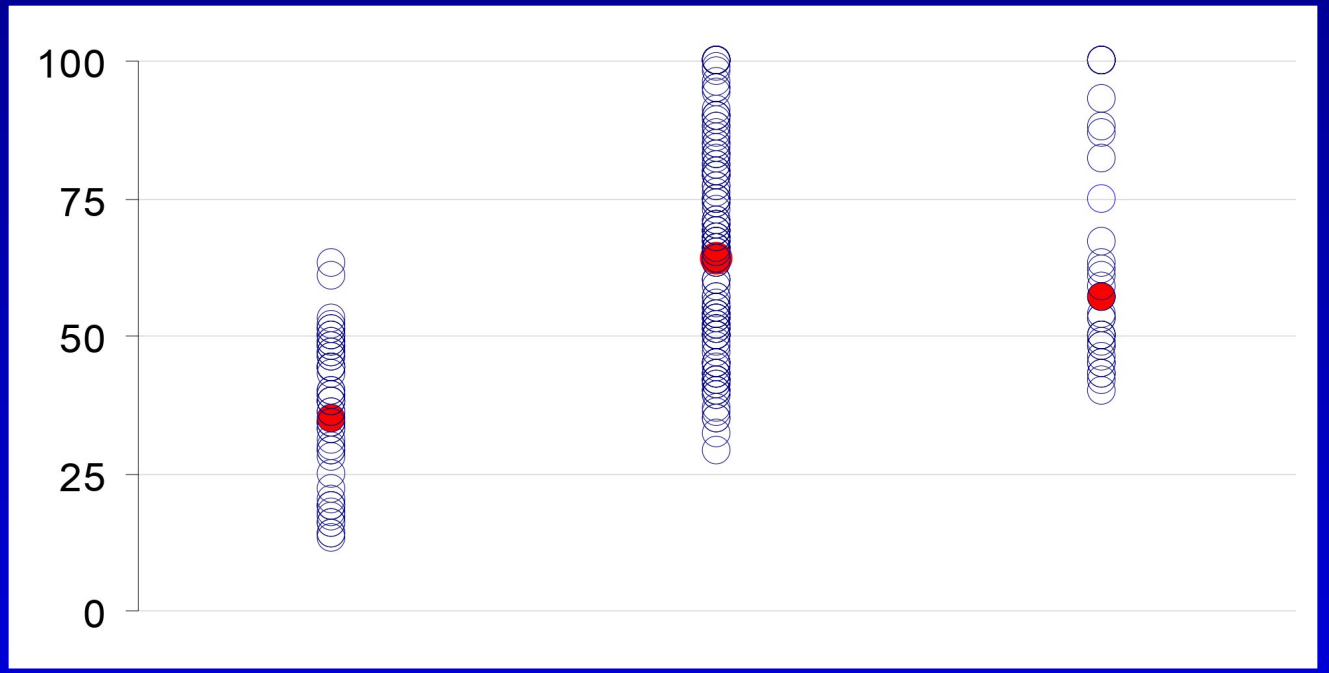
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

A Gain-of-Function Mutation of *JAK2* in Myeloproliferative Disorders

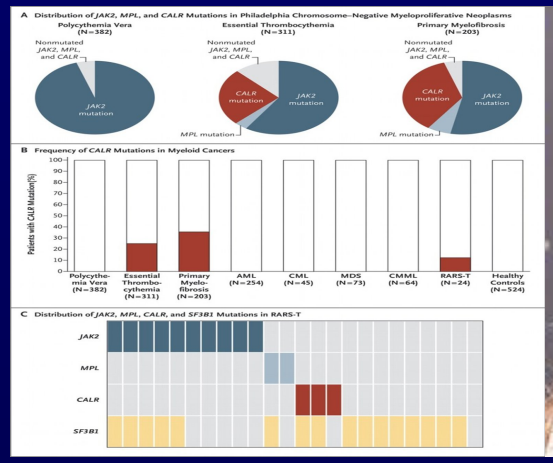
Robert Kralovics, Ph.D., Francesco Passamonti, M.D., Andreas S. Buser, M.D., Soon-Siong Teo, B.S., Ralph Tiedt, Ph.D., Jakob R. Passweg, M.D., Andre Tichelli, M.D., Mario Cazzola, M.D., and Radek C. Skoda, M.D.

Neutrophil JAK2 V617F allele % in MPN



Pemmaraju, Moliterno AR, Williams DM , Rogers 0, Spivak JL, Leukemia 2007 Oct;21(10):2210-2
Stein BL, et al Haematologica 2010
Moliterno AR, et al Exp Hematol 2008

CALR Mutation



- Chromosome 19p13.3
  - Exon 9 of CALR (insertions or deletions)
- Calreticulin = protein ☾ Ca++binding function / Endoplasmic reticulum
- Also found in nucleus; possible role transcription regulation
- Klampfel et al NEJM 2013: CALR in 25% pts with JAK2 negative ET, and in 35% in JAK2 negative MF

Nangalia J et al NEJM: Klampfl T et al. N Engl J Med 2013;369:2379-2390.

MPL Mutation: ET and MF

- MPL = proto-oncogene that encodes for TPOR (hematopoietic growth factor receptor for myeloid stem cells)
- MPL mutations ☾ dimerization of TPOR ☾ actives JAK2 and the TPO pathway

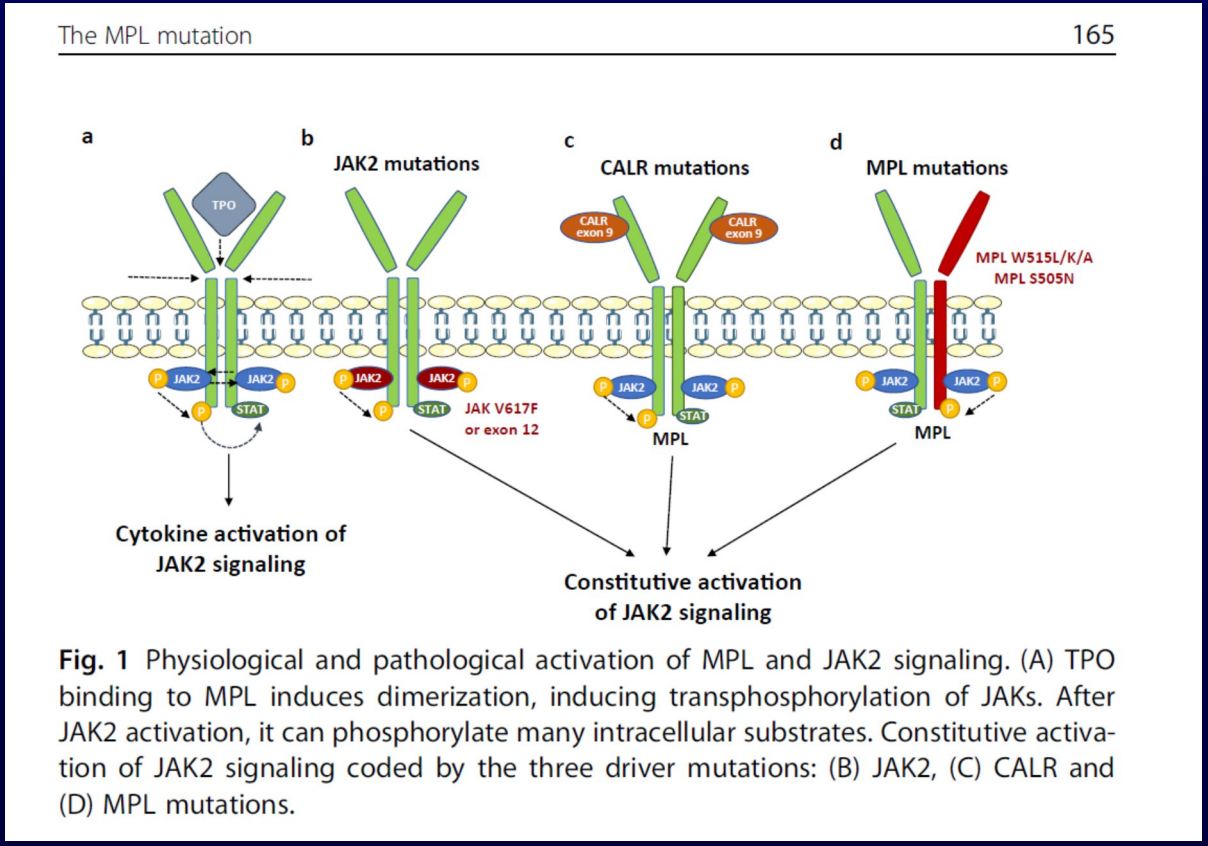
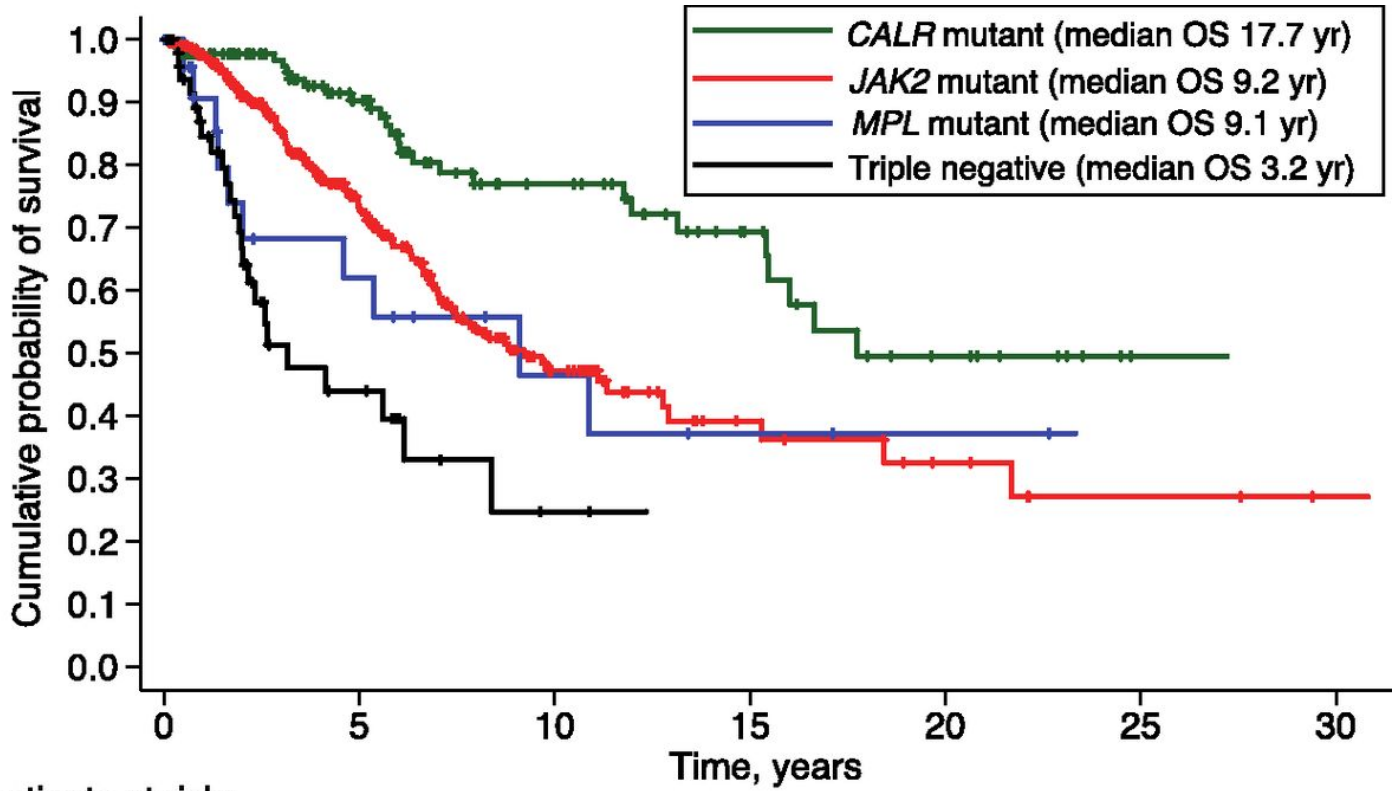


Fig. 1 Physiological and pathological activation of MPL and JAK2 signaling. (A) TPO binding to MPL induces dimerization, inducing transphosphorylation of JAKs. After JAK2 activation, it can phosphorylate many intracellular substrates. Constitutive activation of JAK2 signaling coded by the three driver mutations: (B) JAK2, (C) CALR and (D) MPL mutations.

Guglielmelli P and Calabresi L : The MPL Mutation, Chapter Five, Volume 365, 2021, pages 163-178

Kaplan-Meier analysis of survival of PMF patients stratified according to their driver mutation.



No. of patients at risk:

CALR mutant	140	72	37	19	9	1
JAK2 mutant	396	135	39	13	7	3
MPL mutant	25	10	5	3	2	0
Triple negative	53	11	2	0	0	0

Elisa Rumi et al. Blood 2014;124:1062-1069

Myelofibrosis MF: WHO 2016

- **Major criteria:**
- 1. Presence of megakaryocytic proliferation and atypia, accompanied by either reticulin and/or collagen fibrosis grades 2 or 3*

- 2. Not meeting WHO criteria for ET, PV, BCR-ABL1+ CML, myelodysplastic syndromes, or other myeloid neoplasms

- 3. Presence of JAK2, CALR or MPL mutation or in the absence of these mutations, presence of another clonal marker ** or absence of reactive myelofibrosis ***

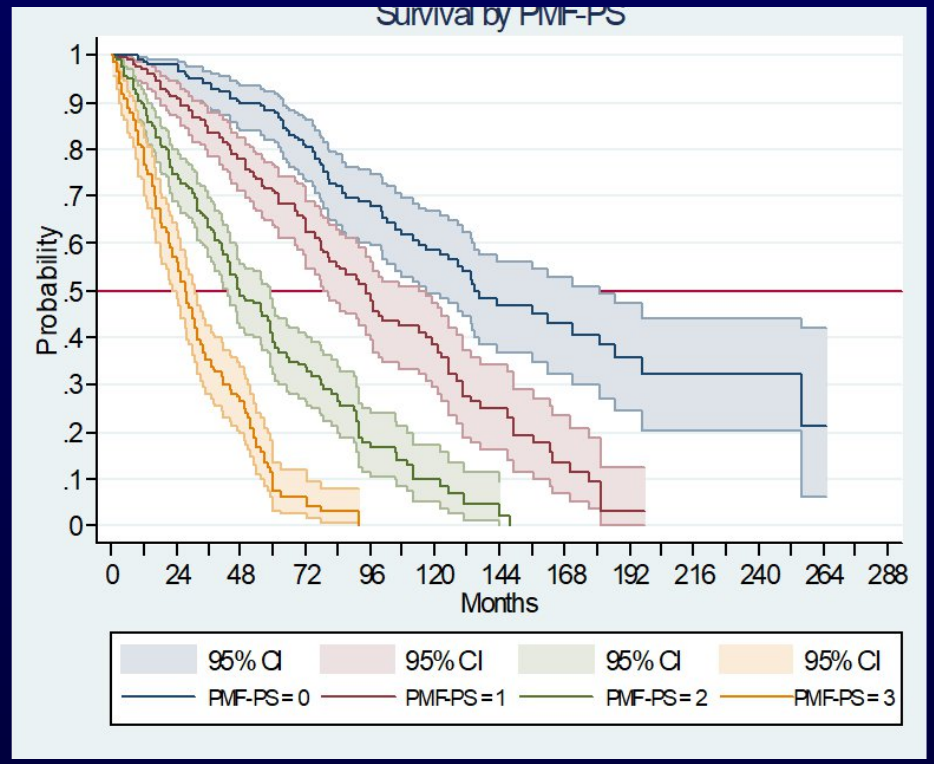
- **Minor criteria: Presence of at least 1 of the following, confirmed in two consecutive determinations:**
- a. Anemia not attributed to a comorbid condition
- b. Leukocytosis >11 x 109 /L
- c. Palpable splenomegaly
- d. LDH increased to above upper normal limit of institutional reference range
- e. Leukoerythroblastosis "tear drop" cells

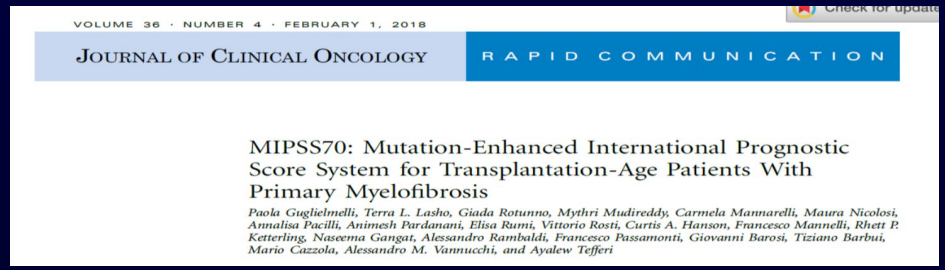
- **Diagnosis of overt PMF requires meeting all three major criteria, and at least one minor criterion** * see Table 8 ** in the absence of any of the 3 major clonal mutations, the search for the most frequent accompanying mutations (e.g.ASXL1, EZH2, TET2, IDH1/IDH2, SRSF2, SF3B1) are of help in determining the clonal nature of the disease
- ***BM fibrosis secondary to infection, autoimmune disorder or other chronic inflammatory conditions, hairy cell leukemia or other lymphoid neoplasm, metastatic malignancy, or toxic (chronic) myelopathies

Arber DA et al WHO 2016 Revision Blood. 2016 May 19;127(20):2391-405

MF: Treatment based on Risk Stratification-Proposal: IPSS, DIPSS, MIPSS

- Low risk: supportive care, transfusions, close surveillance, clinical trials
- Intermediate risk: standard treatment, JAK2 inhibitors, clinical trials, consider allo-SCT
- High risk: JAK2 inhibitors, clinical trials, allo-SCT





MF: Further scoring systems

- **DIPSS (dynamic)—Mayo (Blood 2010;115)**
  - Modified IPSS to be able to calculate over time: all 1 pt except Hb (2 points)
  - Age >65
  - WBC >25K
  - Hb <10: 2 points
  - Circulating blasts greater than or equal to 1%
  - Constitutional sxs

- **DIPSS Plus—adds 3 new factors, each 1 point (Mayo, 2011 JCO)**
  - Unfavorable karyotype
  - Plt count <100K
  - Transfusion need

- **MIPSS /MIPSS-PLUS**
  - BM fibrosis
  - Molecular : CALR ; HR molecular mutations

JAK Inhibitors for Myelofibrosis: 4 Approved Agents

Ruxolitinib (JAK1/2)

- Approved for intermediate-/high-risk MF based in part on COMFORT trials[1]

Fedratinib (JAK2/FLT3)

- Approved for INT-2/high-risk MF; validated by JAKARTA and long-term safety evidence (where no cases of WE were reported)[2,3]

Pacritinib (JAK2/FLT3/IRAK1)

- Approved for adults with adults with intermediate- or high-risk MF with platelets <50 x 10^9/L
- Validated by PERSIST trials[4]

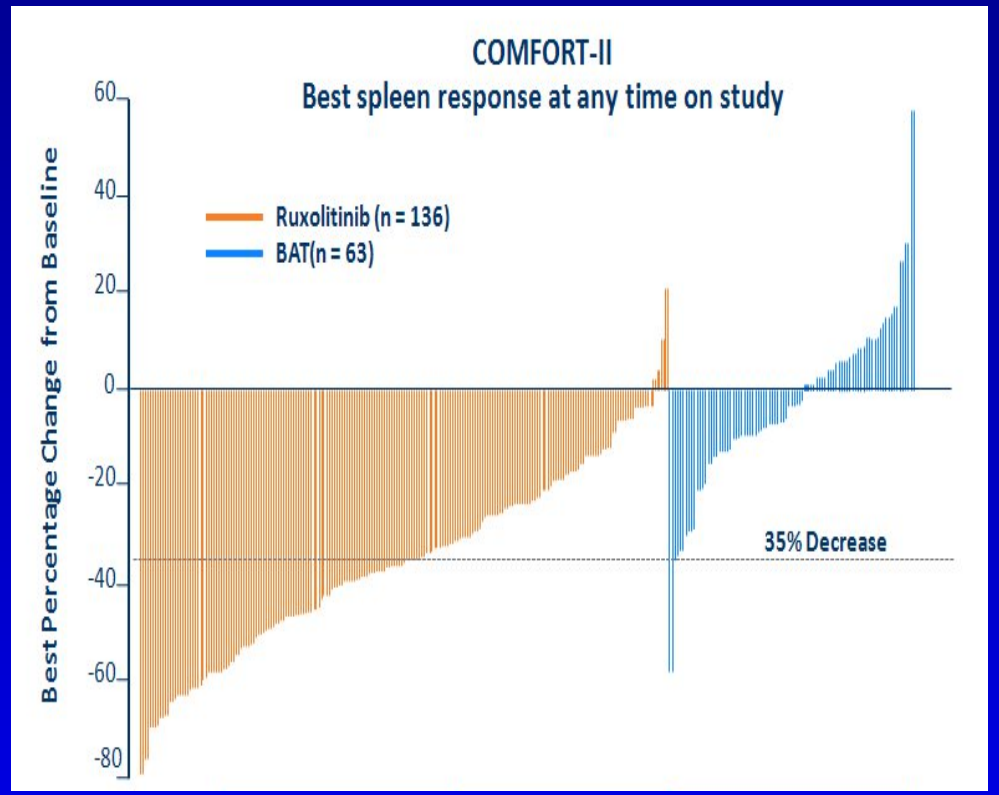
Momelotinib (JAK1/JAK2/ACVR1)

- Approved for intermediate- or high-risk MF patients with anemia
- Validated by MOMENTUM study[5] and subpopulation from the SIMPLIFY-1 trial[6]

1. Harrison C et al. *N Engl J Med*. 2012;366:787-798. 2. Pardanani A et al. *Br J Haematol*. 2021;195:244-248. 3. Pardanani A et al. ASH 2020. Abstract 3006. 4. Mascarenhas J et al. *JAMA Oncol*. 2018;4:652-659. 5. https://clinicaltrials.gov/study/NCT04173494. 6. https://clinicaltrials.gov/ct2/show/NCT01969838.

Ruxolitinib - First-in-Class JAK1/JAK2 inhibitor

- In 2012, Ruxolitinib became first FDA approved drug for MF
- Reduction in spleen size and improvement in symptom burden- based on Ruben Mesa MPN TSS- Total Symptom Burden scale

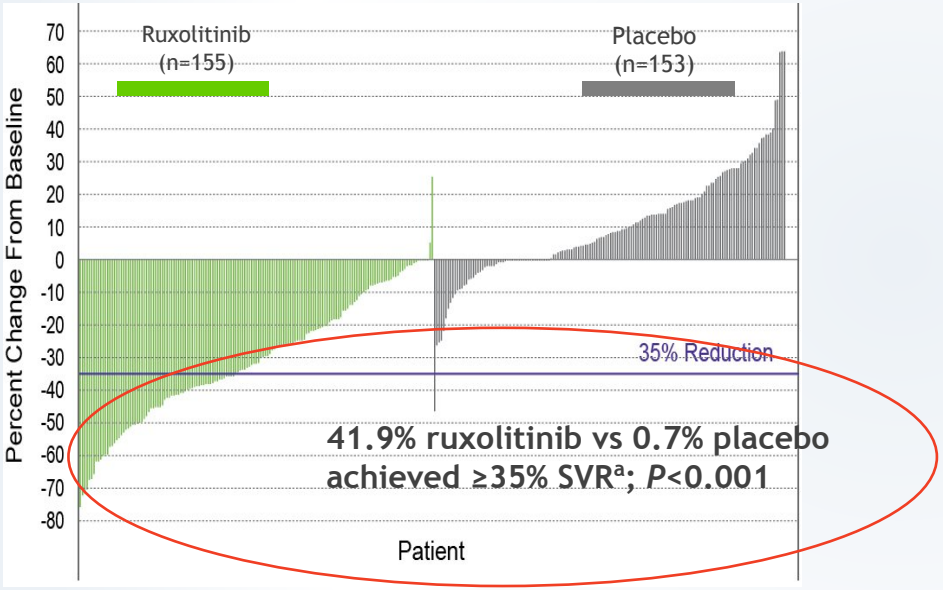


Harrison, C et al NEJM 2012;366:787-798
Verstovsek, S et al NEJM 2012;366:799-807

COMFORT-I: Key Efficacy Endpoints

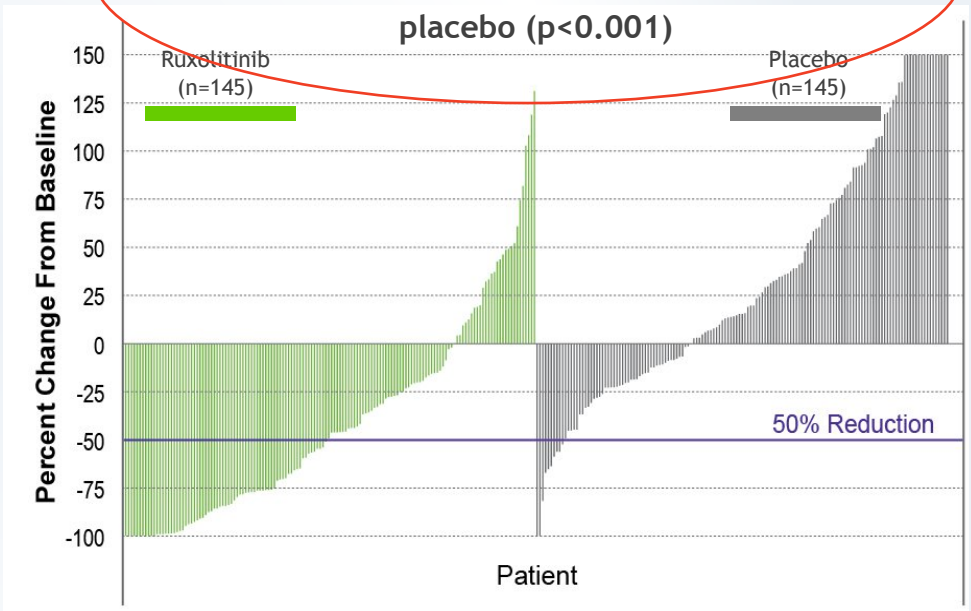
SVR responses were seen with ruxolitinib in JAK2^{V617F}-positive patients and JAK2^{V617F}-negative patients, relative to placebo

Primary Endpoint: ≥35% SVR at 24 Weeks



OR, 134.4 (95% CI: 18.0, 1004.9); *P*<0.001

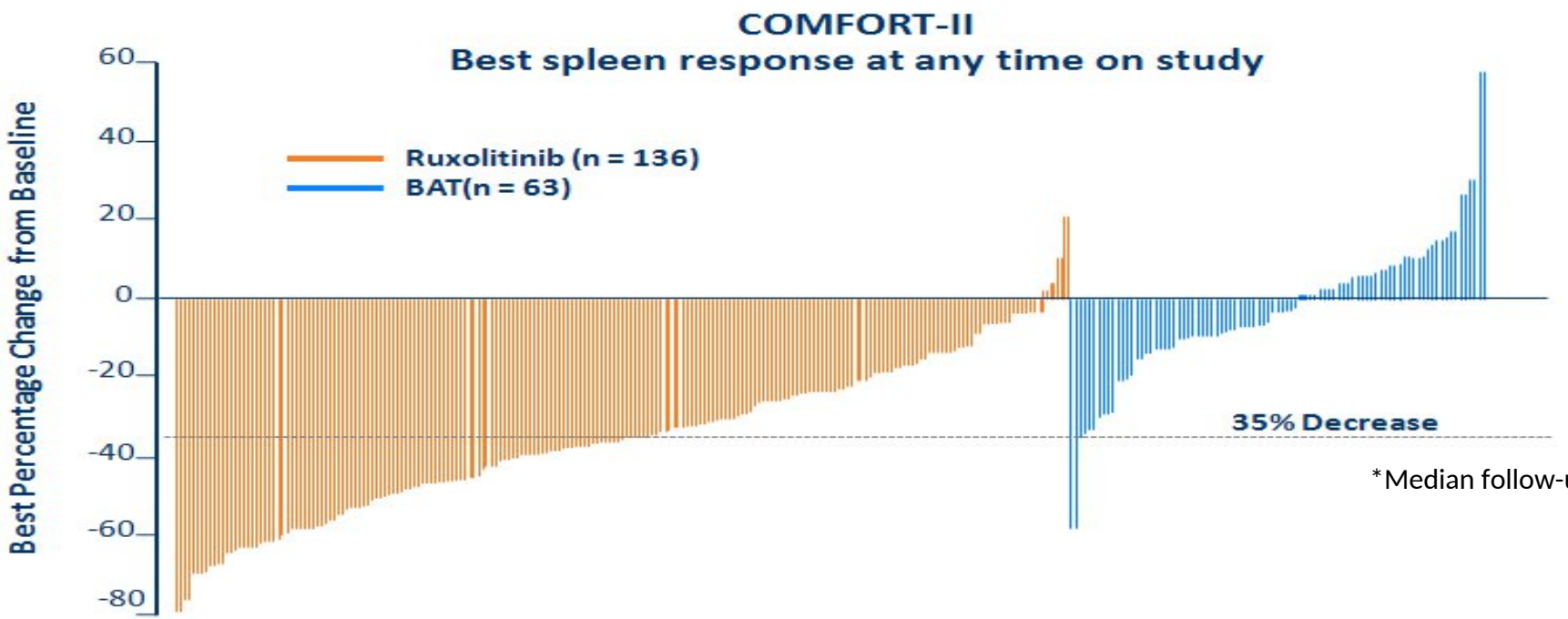
TSS at 24 Weeks: 45.9% in RUXO arm vs 5.3% in placebo (p<0.001)



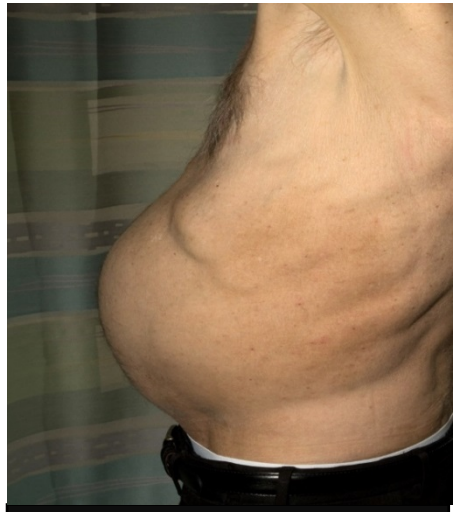
OR, 15.3 (95% CI: 6.9-33.7); *P*<0.001

[a] Changes in palpable spleen length in the ruxolitinib and placebo groups mirrored the changes in spleen volume.
SVR, spleen volume reduction; TSS, total symptom score.
Verstovsek S, et al. *N Engl J Med*. 2012;366:799-807.

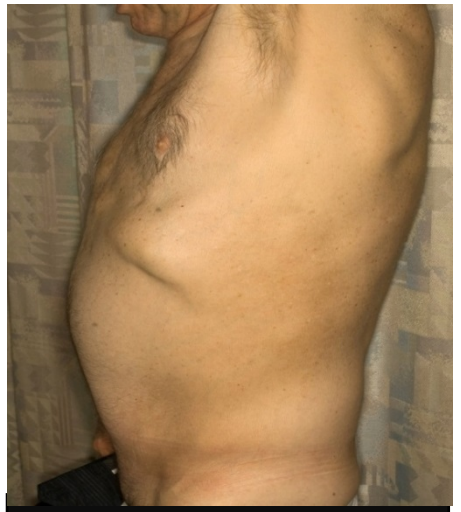
Spleen Volume Response: Ruxolitinib vs. BAT



*Median follow-up: 4.3 years



MF Patient Pre-Ruxolitinib Therapy

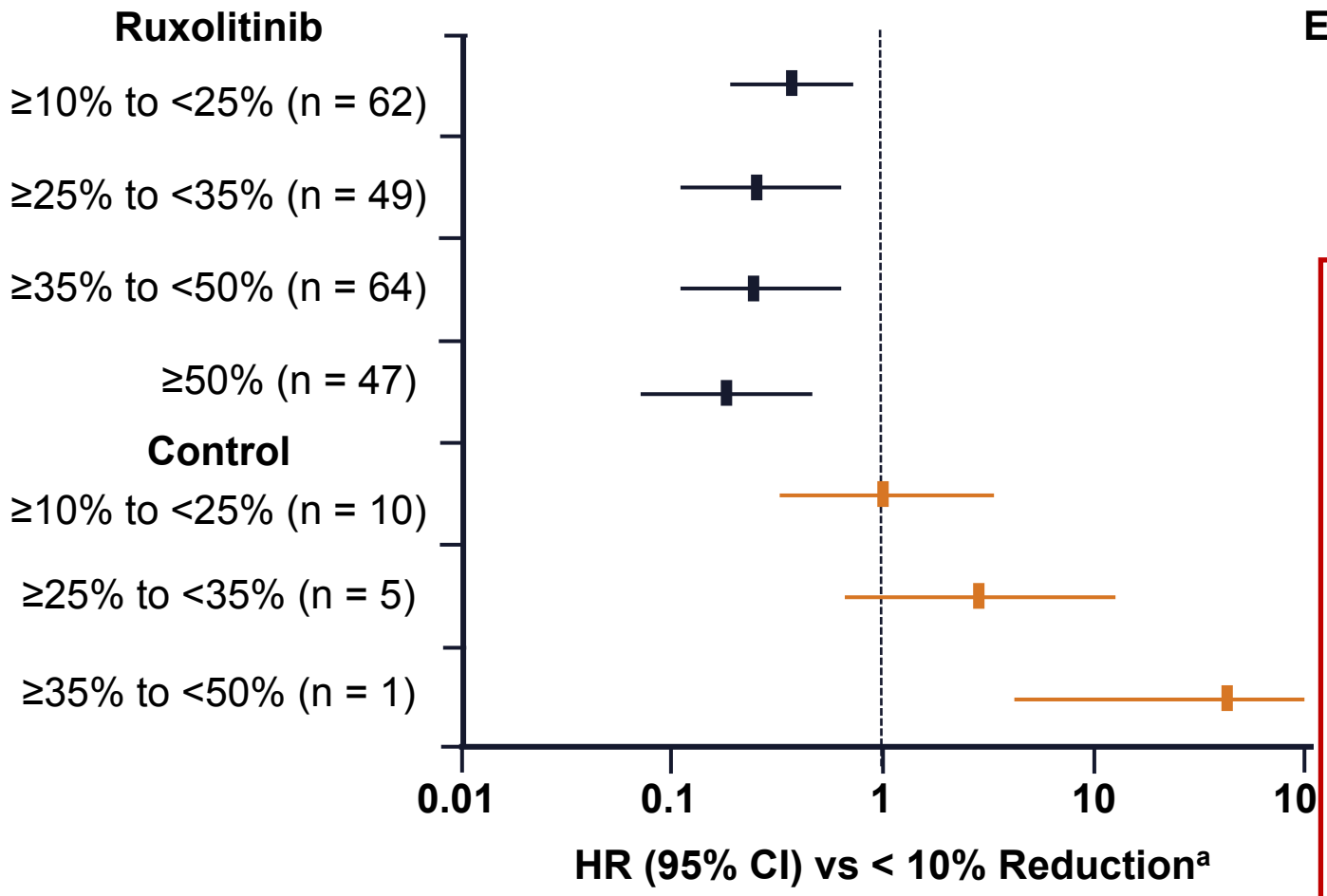


After 2 Months of Therapy

- Dosed based on platelet number (not recommended for platelets <50K)
- It can cause anemia and thrombocytopenia
- Long-term ruxolitinib therapy prolongs survival (earlier intervention and better the spleen response, longer the survival)

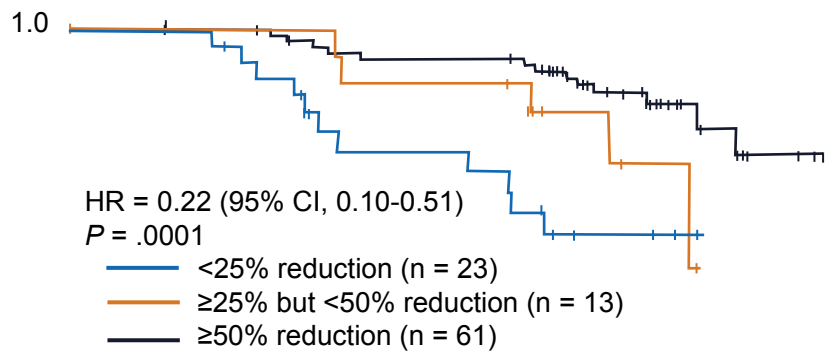
Harrison C, et al. *N Engl J Med*. 2012;366(9):787-798. *Images courtesy of Srdan Verstovsek, MD, PhD*

Better Spleen Response to Ruxolitinib, Better Outcome



Events	HR (95% CI)
15	0.36 (0.18-0.72)
7	0.25 (0.18-0.61

Other evidence: overall survival of patients by degree of spleen length reduction on ruxolitinib

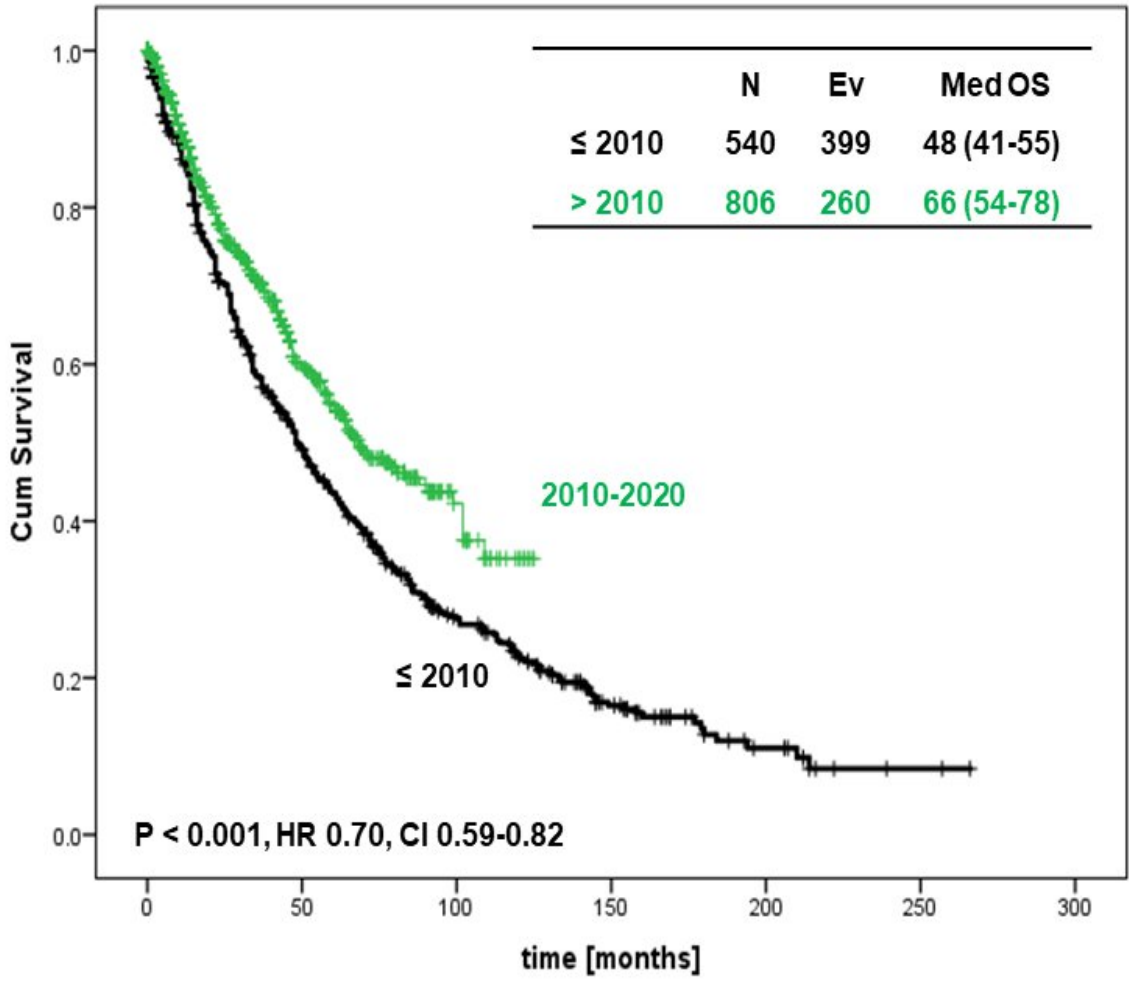


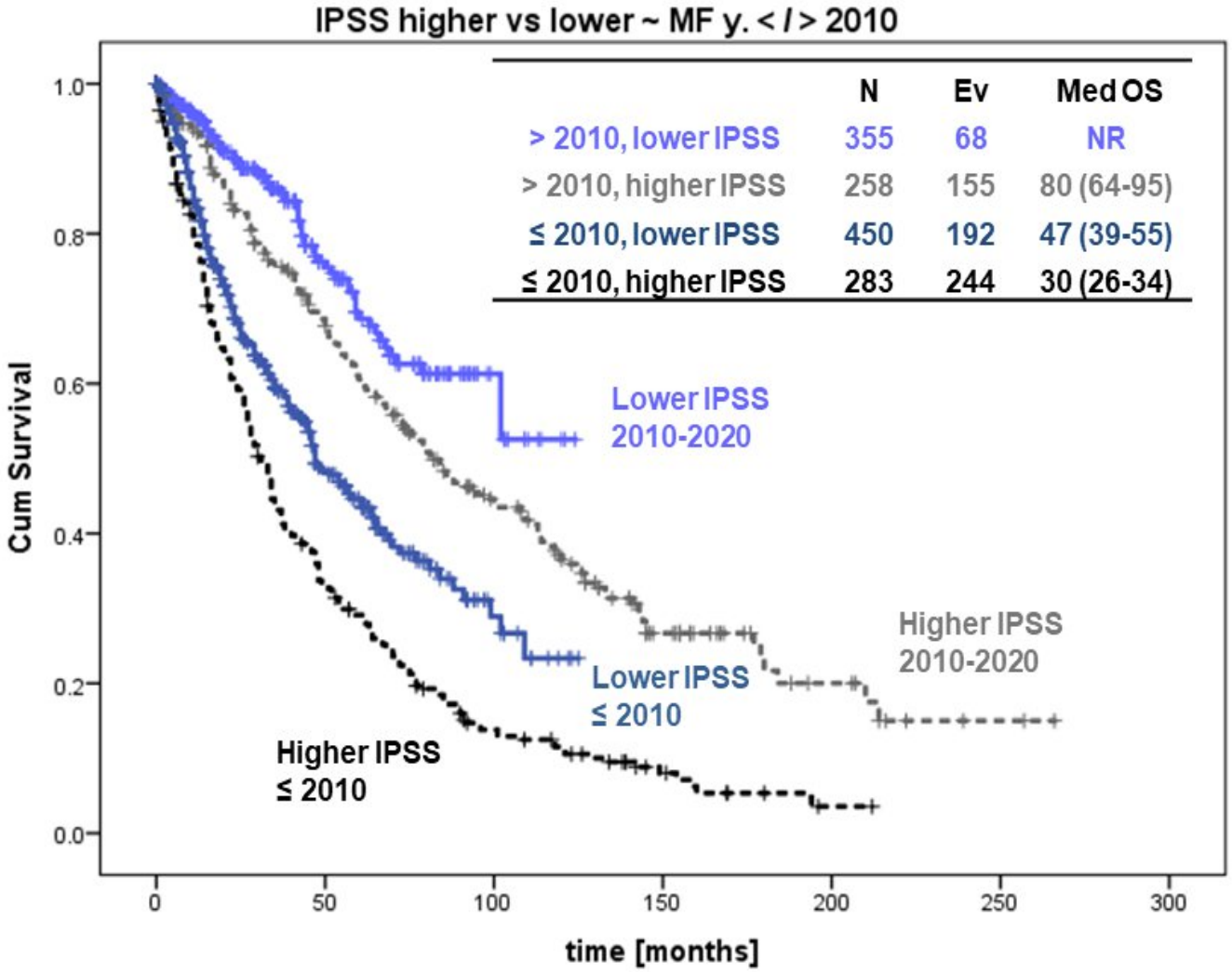
[a]Category includes patients with a <10% reduction from baseline in spleen volume at week 24 or no assessment (ruxolitinib, n=64; control, n=189); among these patients, there were 26 deaths (events) in the pooled ruxolitinib group and 63 deaths in the control group.

1. Vannucchi AM et al. *Haematologica*. 2015;100:1139-1145.

Improved Survival of Patients with Myelofibrosis in the Last Decade

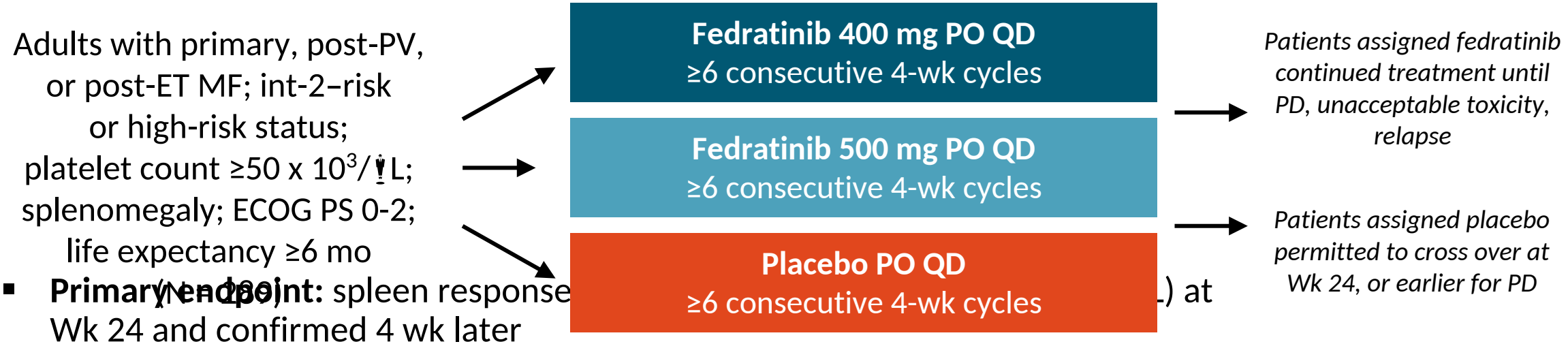
Lucia Masarova, Prithviraj Bose, Naveen Pemmaraju, Lingha Zhou, Sherry Pierce, Zeev Estrov, Hagop Kantarjian, Srdan Verstovsek





JAKARTA: Fedratinib for Primary or Secondary MF

- International, double-blind, randomized phase III trial
  - **Fedratinib**: highly selective, potent inhibitor of wild-type and mutant JAK2; also inhibits FLT3

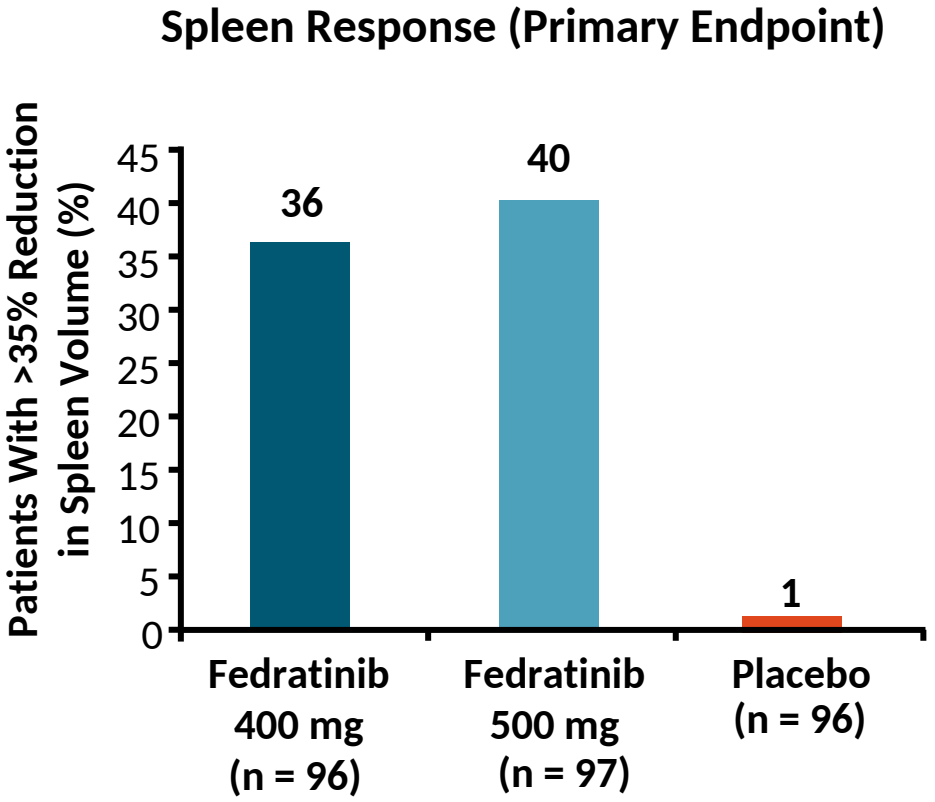


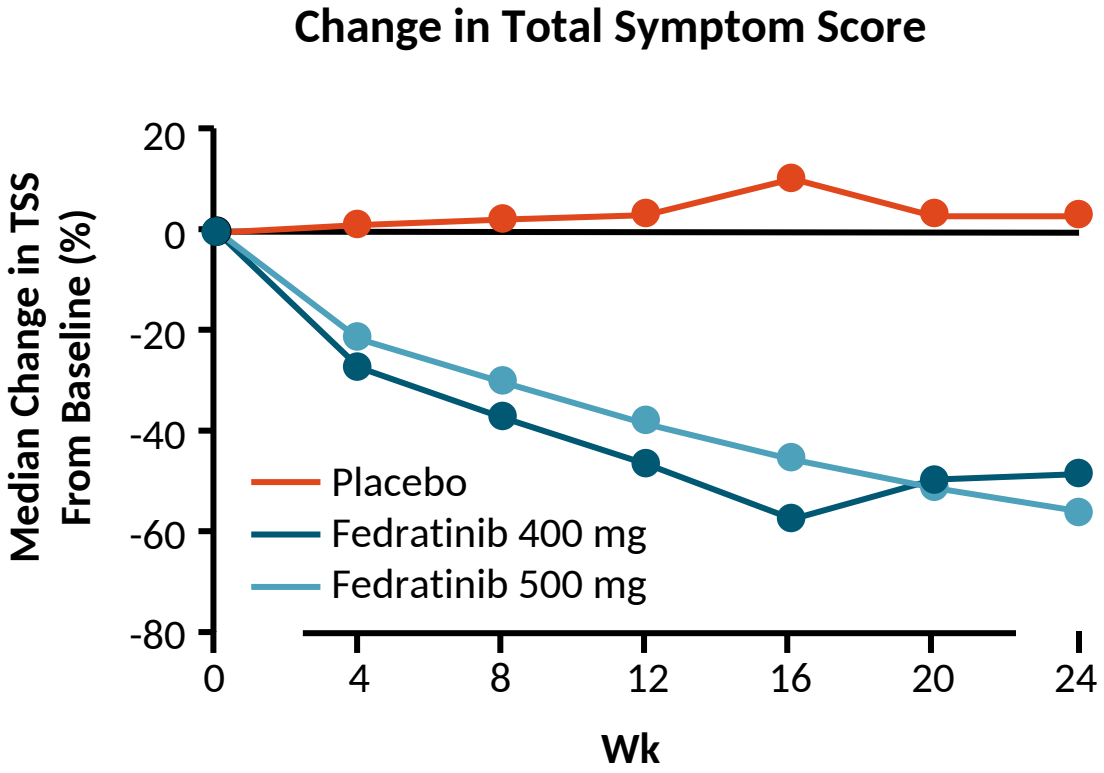
- **Primary endpoint:** spleen response ( ) at Wk 24 and confirmed 4 wk later
- **Secondary endpoints:** symptom response (≥50% reduction in TSS), safety

Pardanani. JAMA Oncol. 2015;1:643.

Slide credit: clinicaloptions.com

JAKARTA: Efficacy





- FDA approved for patients with intermediate-2–risk or high-risk MF who have platelet counts ≥50 × 10^9/L

Pardanani. JAMA Oncol. 2015;1:643. Fedratinib PI.

Slide credit: clinicaloptions.com

Review of Encephalopathy Cases

- Across nine fedratinib trials enrolling 670 MPN or solid tumor patients
- Five potential WE patients
- One subject had malnutrition related to protracted nausea and vomiting, as well as clinical signs and MRI findings consistent with WE
- Two subjects likely experienced WE, both of which recovered without a dose interruption, suggesting fedratinib does not inhibit thiamine absorption
- Two subjects inconclusive or not supportive of WE

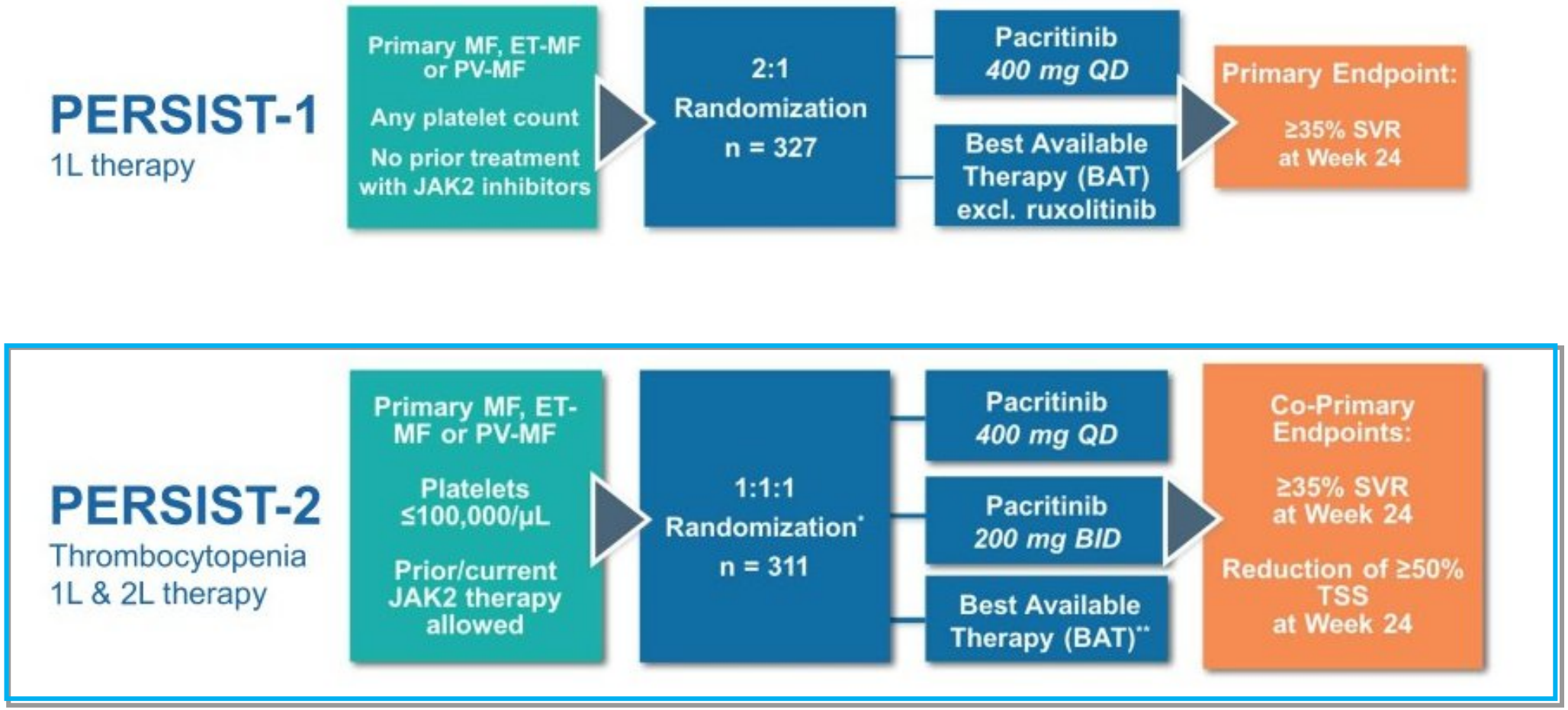
No clear link between WE and fedratinib

1. **Fedratinib does not appear to increase risk for thiamine deficiency beyond its potential to exacerbate malnutrition through poor management of preventable GI events**

2. **Proper management of GI is an important component of care for patients on fedratinib**

1. Harrison CN et al. *Blood*. 2017;130:4197.

Phase 3 PERSIST-1 and PERSIST-2: Study Design



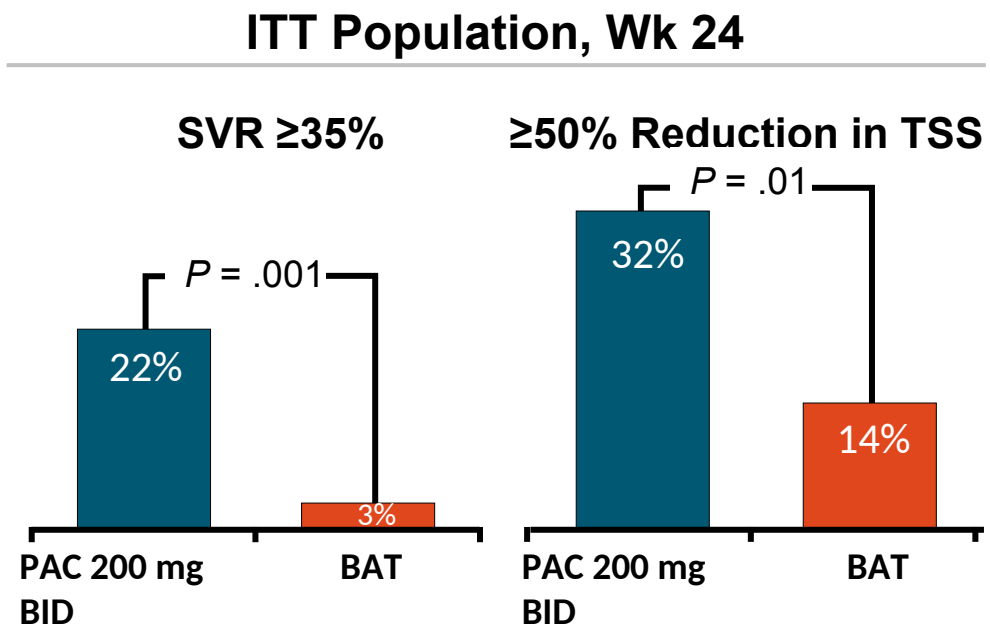
Targeted Oncology | Case-Based ROUNDTABLE

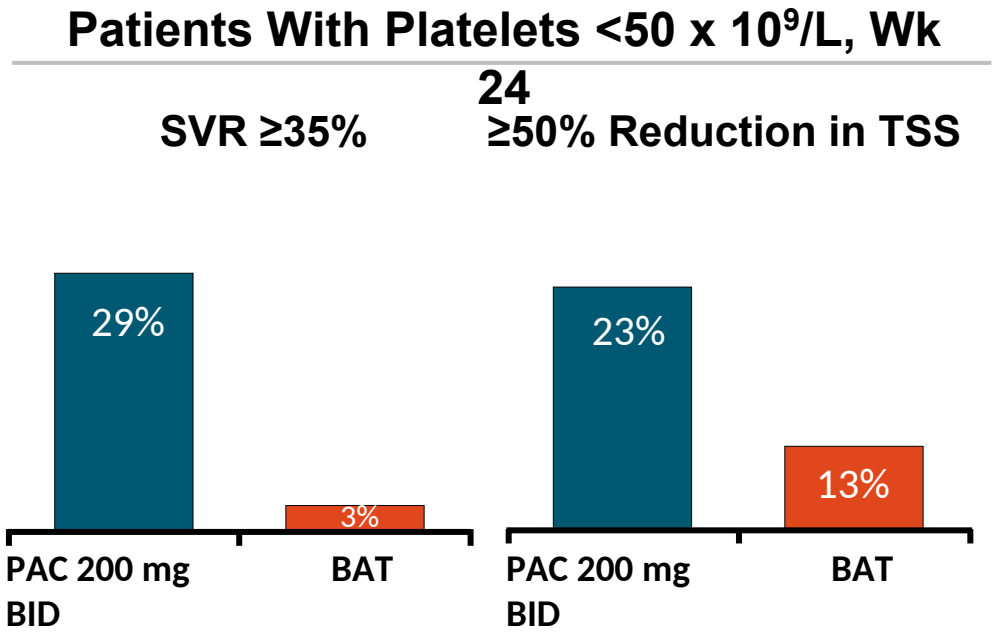
Recommended dosage is 200 mg orally twice daily

** BAT includes ruxolitinib.

Mascarenhas J, et al. *JAMA Oncol*. 2018;4:652-659.

PERSIST-2: Spleen/Symptom Response at 200 mg bid





- Rarely myelosuppressive; can cause GI side effects
- **Pacritinib received accelerated FDA approval as therapy for patients with intermediate/high-risk MF with platelets <50 x 10^9/L**

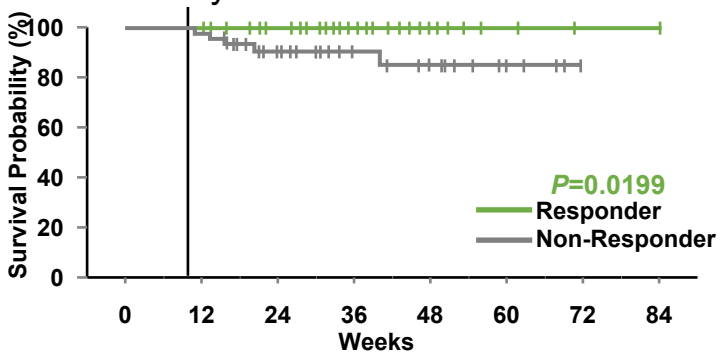
Mascarenhas. JAMA Oncol. 2018;4:652.

Slide credit: clinicaloptions.com

SVR Predicts Survival In MF Patients On Pacritinib But Not Best Available Therapy: Persist-2 Landmark Overall Survival Analysis

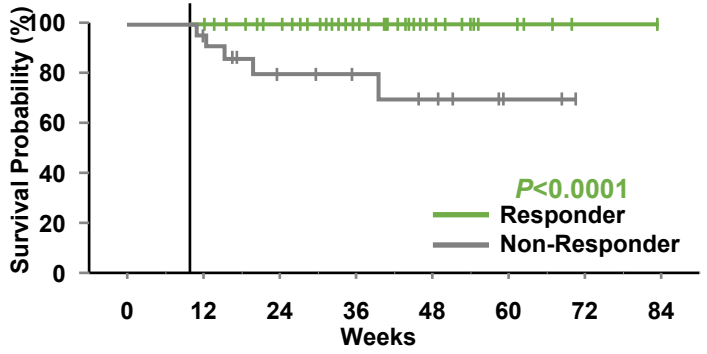
Pacritinib

(B) OS Stratified by **≥20% SVR**



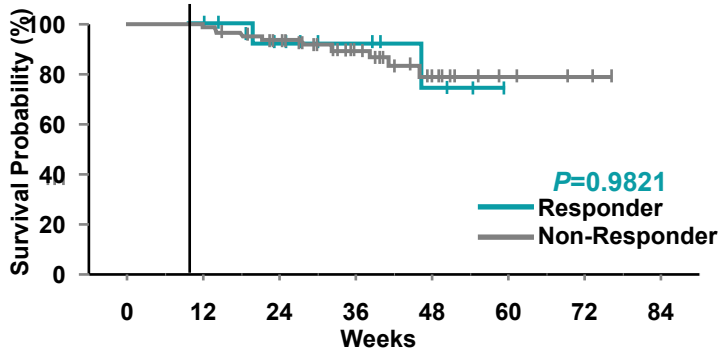
N							
Responder	43	34	25	12	4	1	0
Non-Responder	46	28	16	11	5	0	0

(C) OS Stratified by **≥10% SVR**

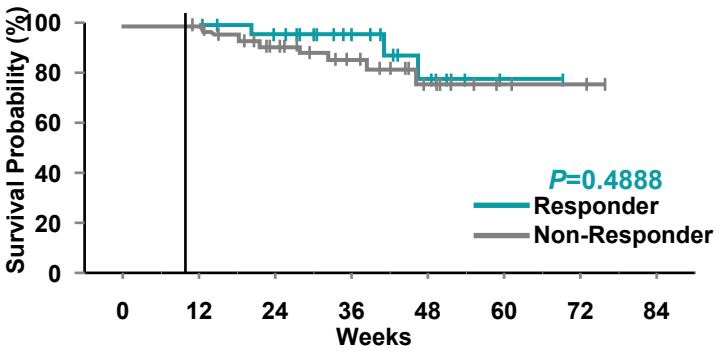


N							
Responder	65	51	33	17	6	1	0
Non-Responder	24	11	8	6	3	0	0

BAT (including RUX)



N							
Responder	15	10	7	3	0	0	0
Non-Responder	69	50	34	15	4	2	0



N							
Responder	28	23	16	7	1	0	0
Non-Responder	58	37	25	11	3	2	0

BAT, best available therapy; MF, myelofibrosis; OS, overall survival; RUX, ruxolitinib; SVR, spleen volume reduction.
Bewersdorf J. *EHA* 2023. #P20010.

DOI: 10.1002/jha2.591

eJHaem

SHORT REPORT

Risk-adjusted safety analysis of the oral JAK2/IRAK1 inhibitor pacritinib in patients with myelofibrosis

Naveen Pemmaraju[1] | Claire Harrison[2] | Vikas Gupta[3] | Srdan Verstovsek[1] | Bart Scott[4] | Stephen T. Oh[5] | Francesca Palandri[6] | Haifa Kathrin Al-Ali[7] | Marta Sobas[8] | Mary Frances McMullin[9] | Ruben Mesa[10] | Sarah Buckley[11] | Karisse Roman-Torres[11] | Alessandro Vannucchi[12] | Abdulraheem Yacoub[13]

Abstract

The safety profile of the novel oral JAK2/IRAK1 inhibitor pacritinib in patients with cytopenic myelofibrosis was described in the Phase 2 PAC203 and Phase 3 PERSIST-2 studies. To account for longer treatment durations on the pacritinib arms compared to best available therapy (BAT), we present a risk-adjusted safety analysis of event rates accounting for different time on treatment. While the rate of overall events was higher on pacritinib compared to BAT, the rate of fatal events was lower, and there was no excess in bleeding, cardiac events, secondary malignancy, or thrombosis on pacritinib, including in patients with severe thrombocytopenia.

TABLE 1 Baseline patient and disease characteristics

	PAC203	PERSIST-2			Pooled Analysis
Characteristic	PAC 200 mg BID *n*= 54	PAC 200 mg BID *n* = 106	BAT *n* = 98	BAT = RUX *n* = 44	PAC 200 mg BID *n* = 160
Age (years), median (range)	69 (37, 85)	67 (39, 85)	68 (32, 83)	68 (42, 83)	68 (37, 85)
Female gender, *n* (%)	22 (41%)	44 (42%)	45 (46%)	15 (34%)	66 (41%)
ECOG PS ≥2, *n* (%)	8 (15%)	12 (11%)	18 (18%)	10 (23%)	20 (13%)
PLT ($\times 10^9$/L), median (IQR)[1]	59 (29, 91)	55 (36, 93)	57 (29, 81)	61 (35, 91)	57 (33, 93)
PLT < 50 × 10^9/L, *n* (%)[1]	24 (44%)	47 (44%)	42 (43%)	17 (39%)	71(44%)
HB < 10 g/dl, *n* (%)	41 (76%)	62 (59%)	54 (55%)	23 (52%)	103 (64%)
Receives RBC transfusions, *n* (%)[2]	34 (63%)	49 (46%)	47 (48%)	19 (43%)	83 (52%)
Peripheral blasts ≥1%, *n* (%)	32 (59%)	48 (45%)	46 (47%)	27 (61%)	80 (50%)
Primary MF, *n* (%)	37 (69%)	82 (77%)	60 (61%)	22 (50%)	119 (74%)
DIPSS high risk, *n* (%)	14 (26%)	29 (27%)	26 (27%)	12 (27%)	43 (27%)
Prior JAKi exposure, *n*	54 (100%)	51 (48%)	52 (53%)	32 (73%)	105 (66%)

Pemmaraju N et al eJHaem 2022; 3:1346-1351

MOMENTUM Study Design

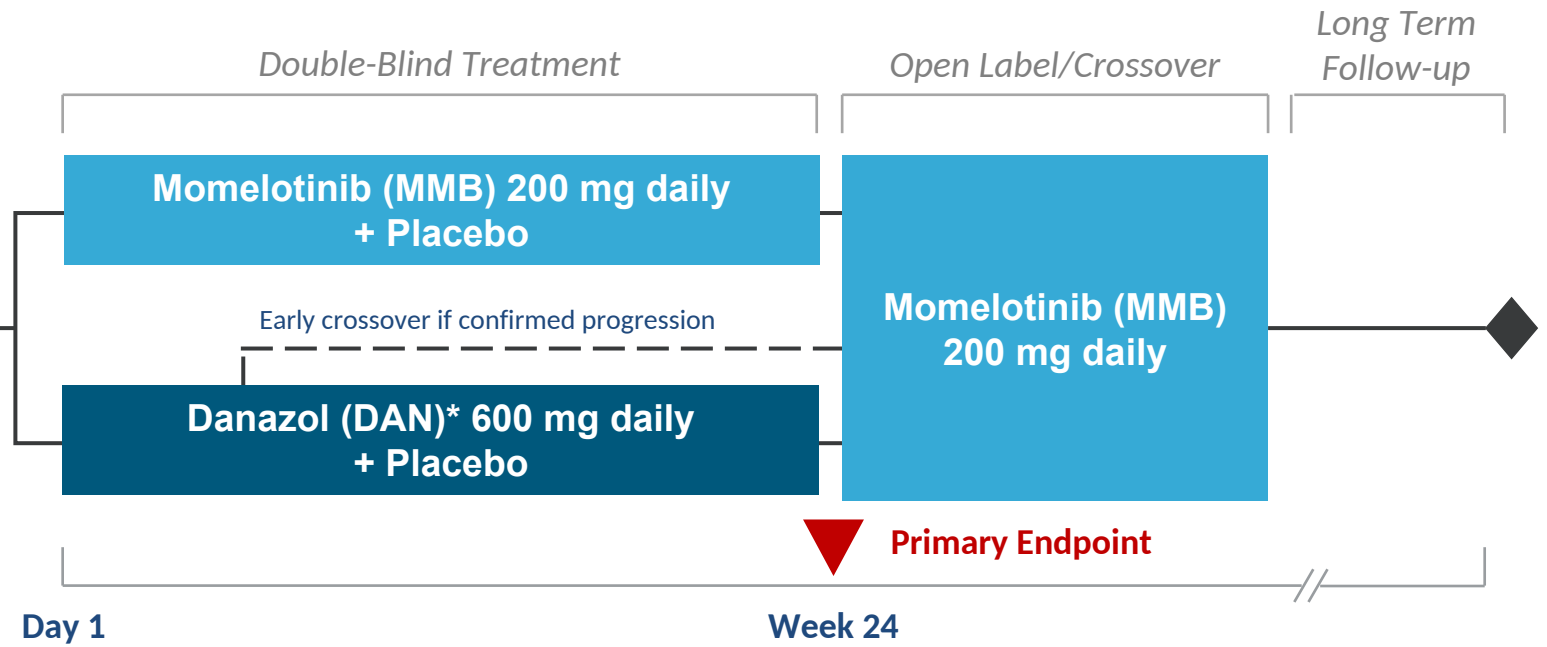
Previously Treated with JAK inhibitor
Symptomatic (TSS ≥10) and Anemic (Hgb <10 g/dL)

Patients
N=195

2:1 randomization
JAKi taper/washout ≥21 days

Stratification:
- Total symptom score
- Palpable spleen length
- Transfused units in prior 8 wks
- Study site

Planned enrollment: 180
FPE April 2020
LPE June 2021
Database lock Dec 2021



Primary Endpoint

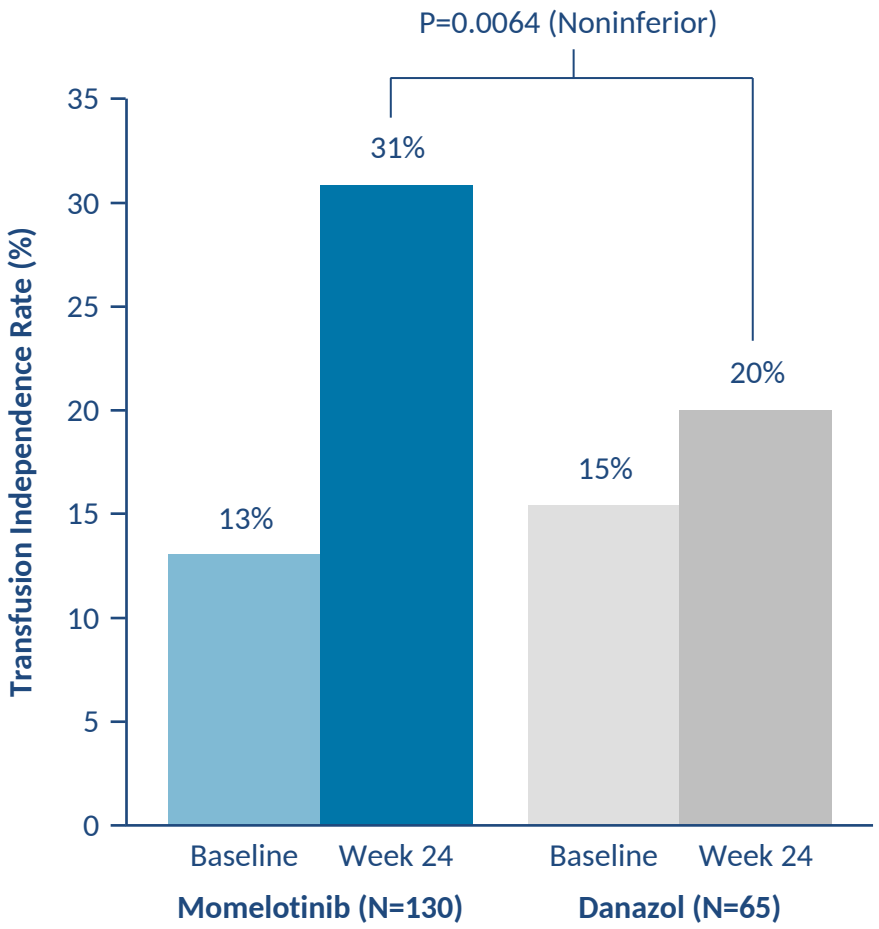
- Total symptom score (TSS) response rate at Week 24

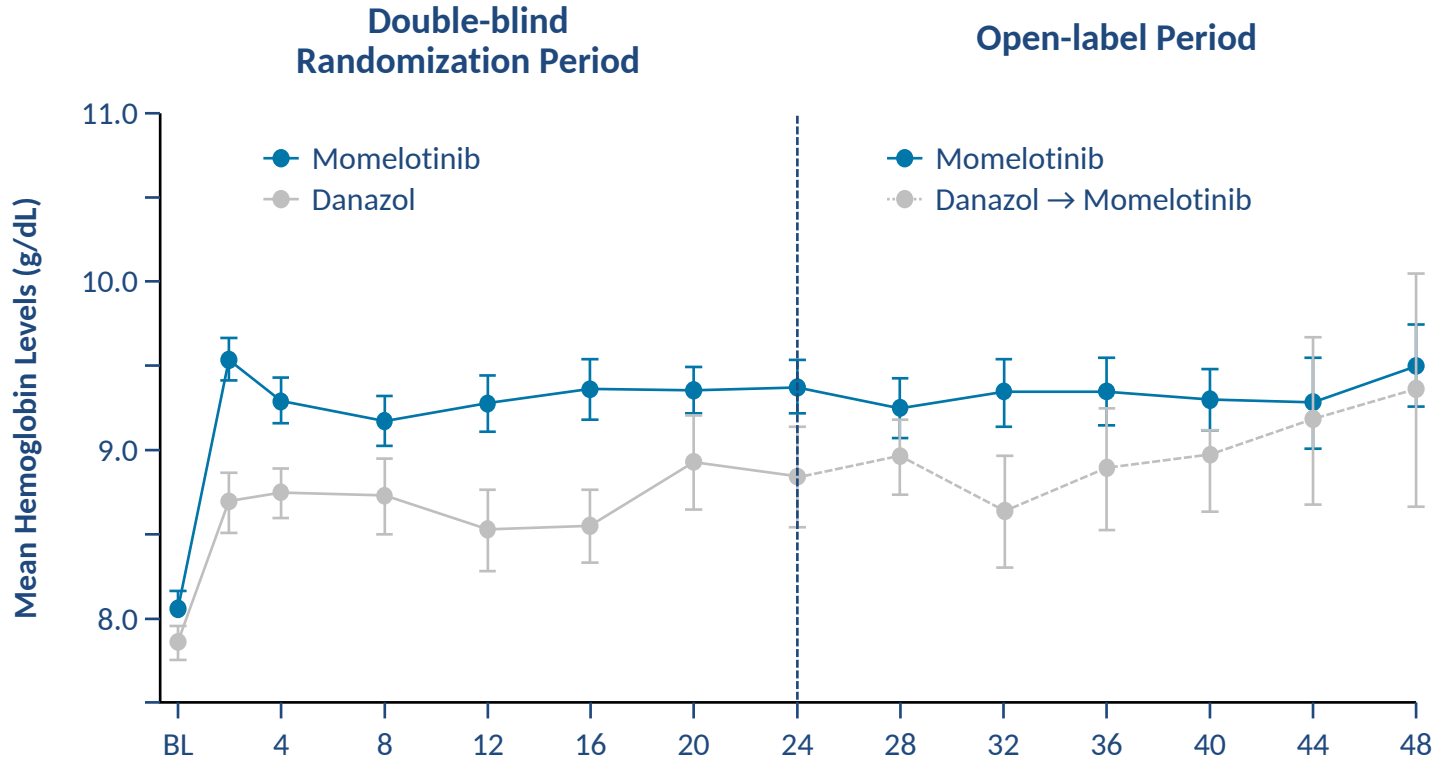
Key Secondary Endpoints

- Transfusion independence (TI) rate at Week 24
- Splenic response rate (SRR) at Week 24

ClinicalTrials.gov: **NCT04173494**
*Danazol was selected as an appropriate comparator given its use to ameliorate anemia in MF patients, as recommended by NCCN, ESMO guidelines.
TSS response defined as achieving ≥50% reduction in TSS over the 28 days immediately prior to the end of week 24 compared to baseline.
TI defined as not requiring red blood cell transfusion in the last 12 weeks of the 24-week randomized period, with all hemoglobin levels during the 12-week interval of ≥8 g/dL.
SRR defined as achieving a **≥25%** or **≥35%** reduction in spleen volume from baseline.

Transfusion Independence* Rate at W24 and Mean Hemoglobin Over Time

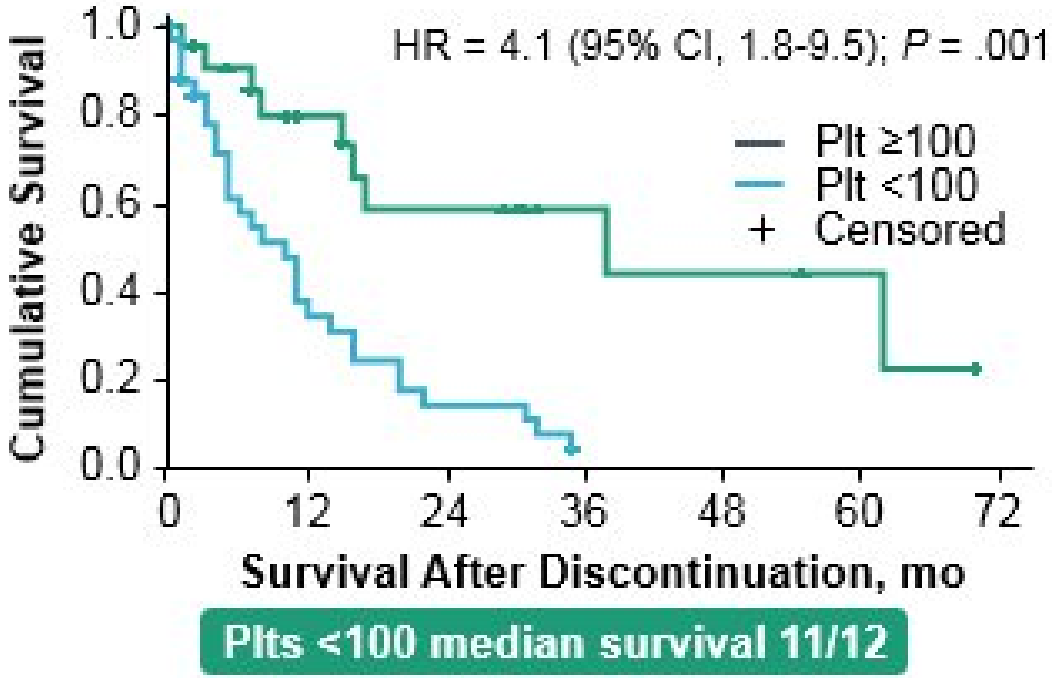




No. of Patients													
Momelotinib	129	105	112	93	87	88	83	67	56	44	42	36	28
Danazol	65	54	50	38	36	36	29	31	21	19	18	13	7

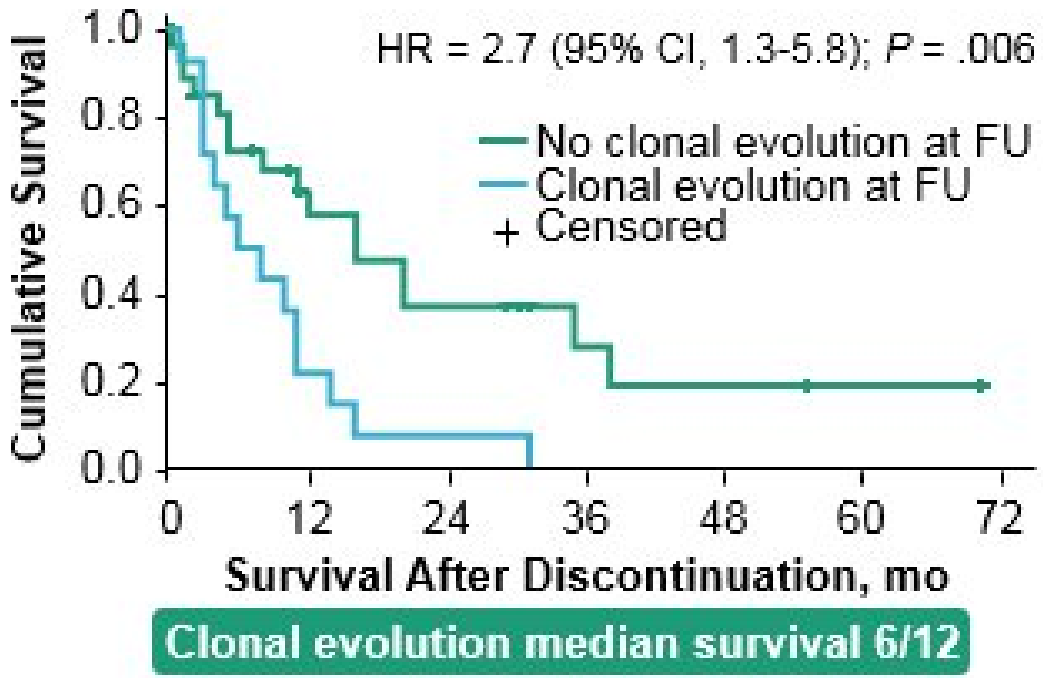
*Defined as not requiring red blood cell transfusion in the terminal 12 weeks of the 24-week randomized period, with all hemoglobin levels during the 12-week interval of ≥8 g/dL.

Prognosis After Ruxolitinib Discontinuation



Plts <100 median survival 11/12

No. at risk							
Plt ≥100	23	12	7	4	3	1	0
Plt <100	33	10	4	0			

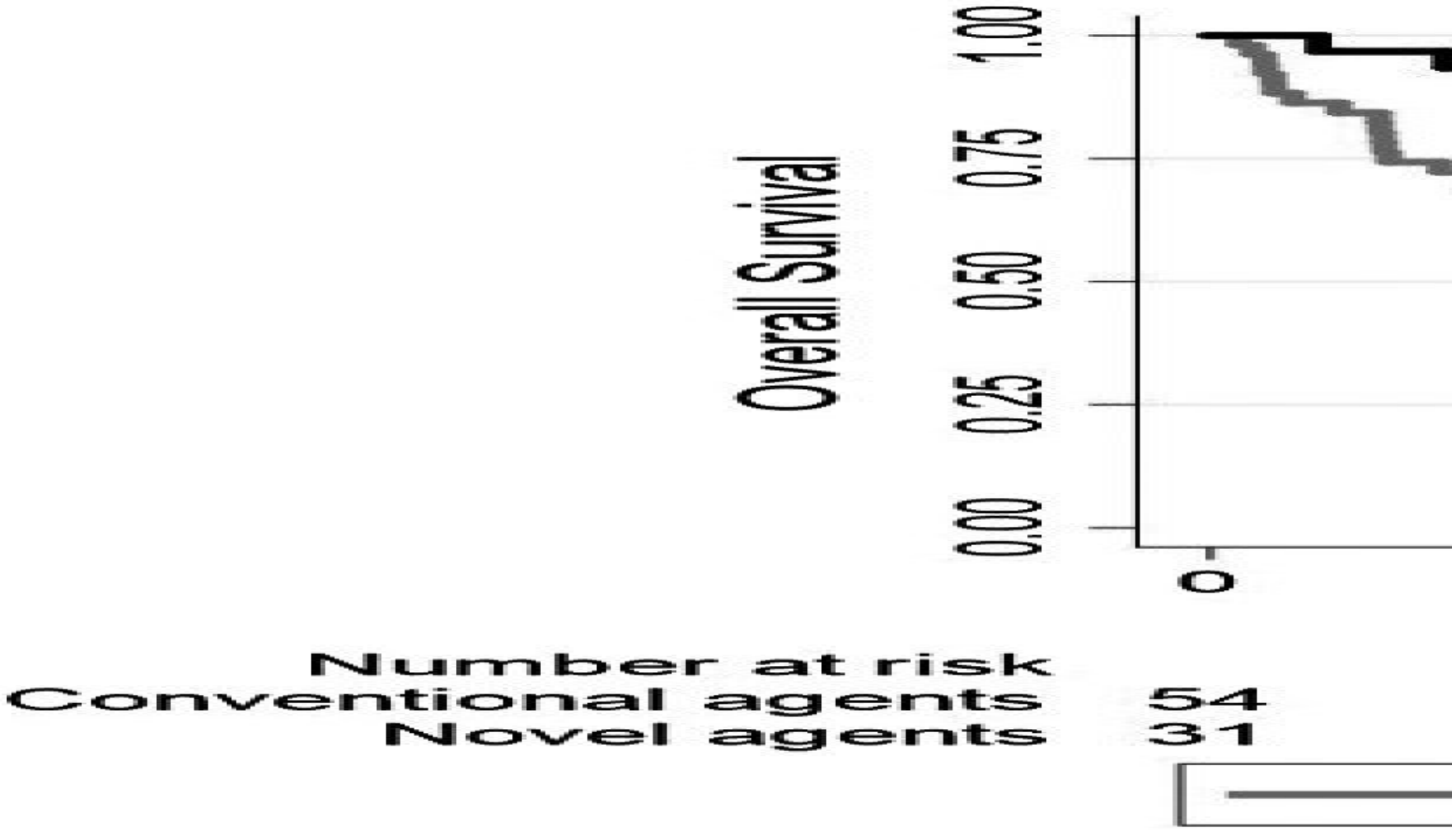


Clonal evolution median survival 6/12

No. at risk							
No CE	28	16	4	4	2	1	0
CE	14	3	3	1	0		

Newberry KJ-Verstovsek S et al. *Blood*. 2017 130:1125-1131.

Life after ruxolitinib: Reasons for discontinuation, impact of disease phase, and outcomes in 218 patients with myelofibrosis



- At 3 years, 41% of patients stopped taking RUXO
- Baseline predictors for RUXO d/c: 1)int-2/HR MF; plt <100; tx-dep; unfavorable karyotype
- n-=55 (19%)patients died while taking RUXO
- Reasons for RUXO d/c: lack of response (23%); loss of spleen response (12%); ruxo-related AE's (27.5%); progression to BP (23%); unrelated to ruxo AE's (9%) & alloSCT in response (5%)
- Med OS s/p RUXO d/c= 13.2 mo
- The use of investigational agents was associated with improved outcomes vs conventional agents

Palandri et al , Cancer, Volume: 126, Issue: 6, Pages: 1243-1252, First published: 20 December 2019, DOI: (10.1002/cncr.32664)

Defining disease modification in myelofibrosis in the era of targeted therapy

Naveen Pemmaraju, MD [1]; Srdan Verstovsek, MD, PhD[1]; Ruben Mesa, MD, FACP [2]; Vikas Gupta, MD[3]; Jacqueline S. Garcia, MD[4]; Joseph M. Scandura, MD [5]; Stephen T. Oh, MD[6]; Francesco Passamonti, MD[7]; Konstanze Döhner, MD[8]; and Adam J. Mead, MD[9]

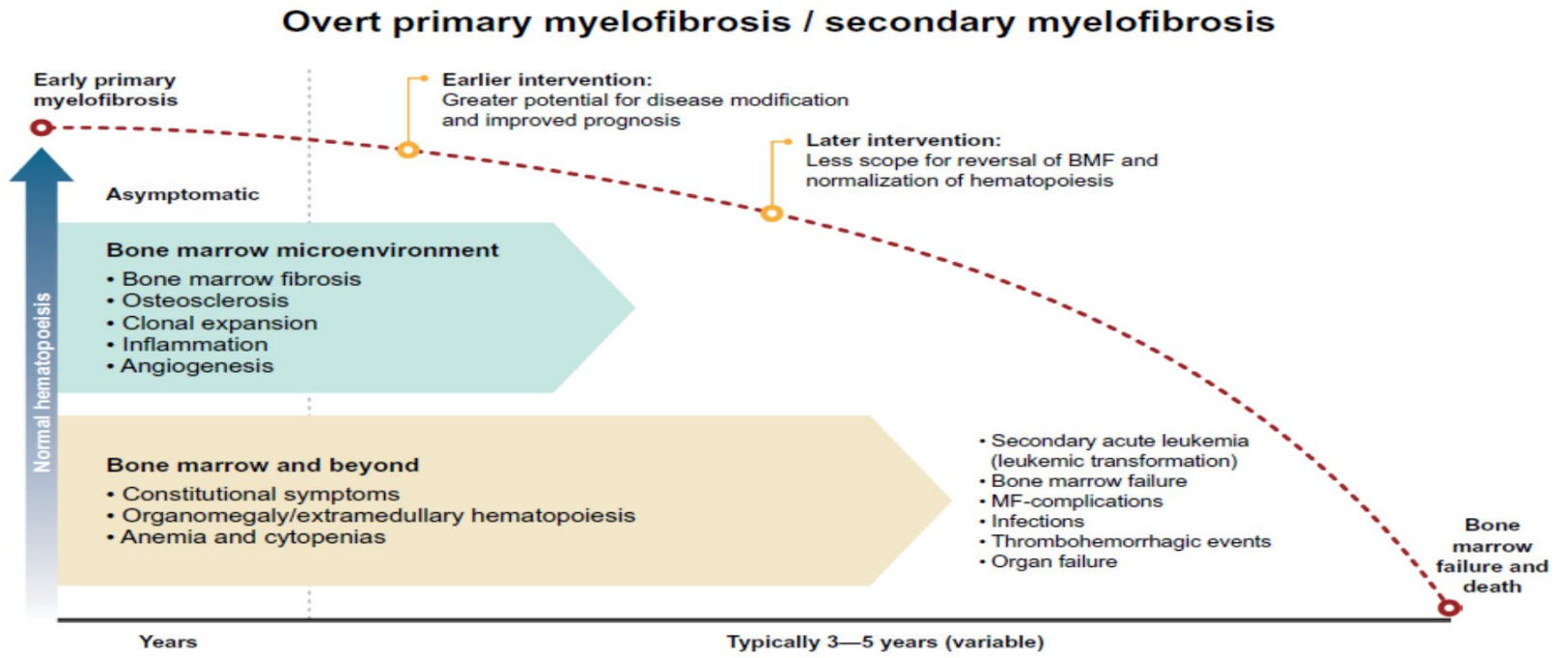
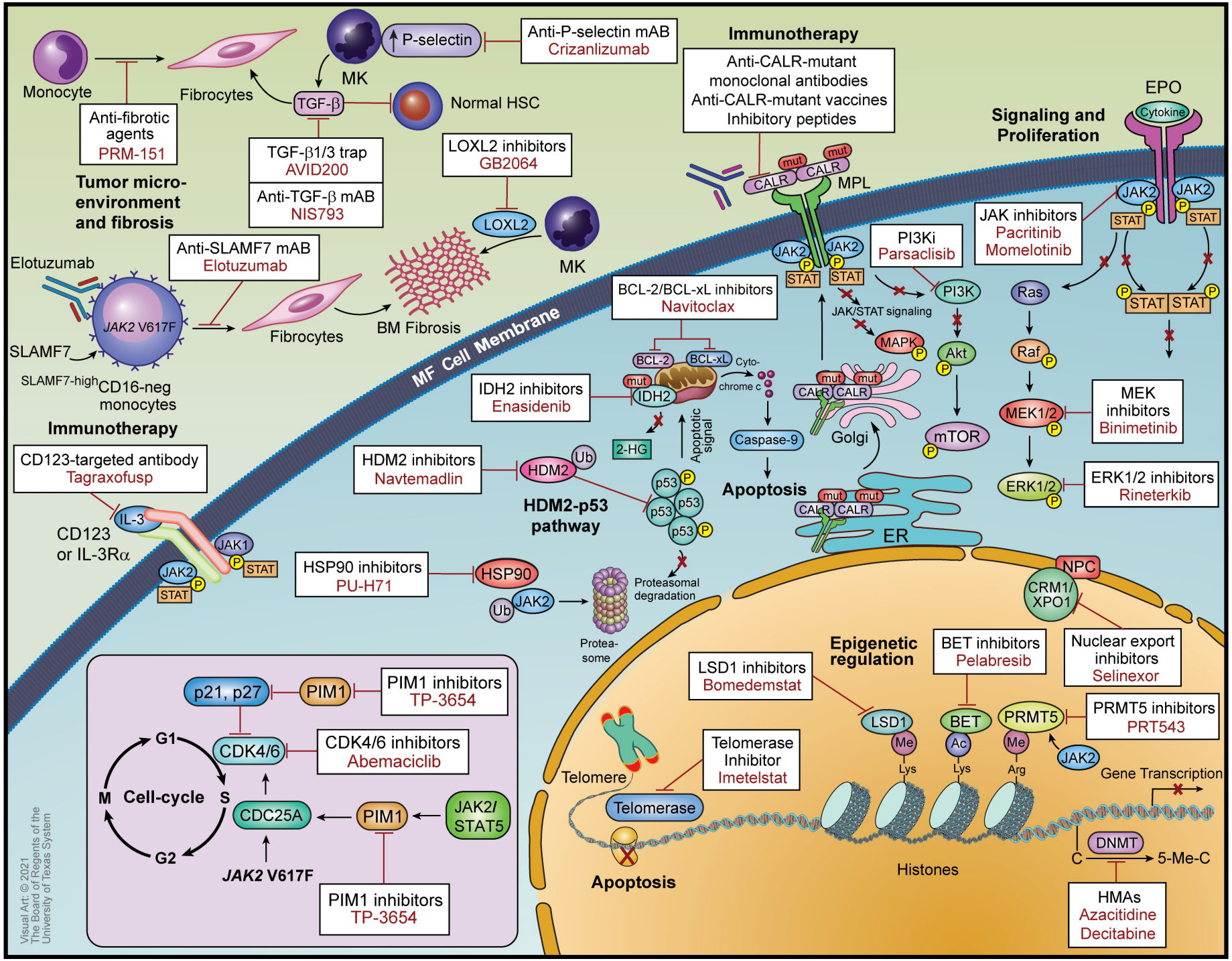


FIGURE 2. Natural history of myelofibrosis and potential time points for intervention. The red dotted line represents the decline in normal hematopoiesis along the natural course of disease.[1,2,23,79] BMF indicates bone marrow fibrosis; MF, myelofibrosis.

Pemmaraju N et al Cancer 2022; 128:2420-2432

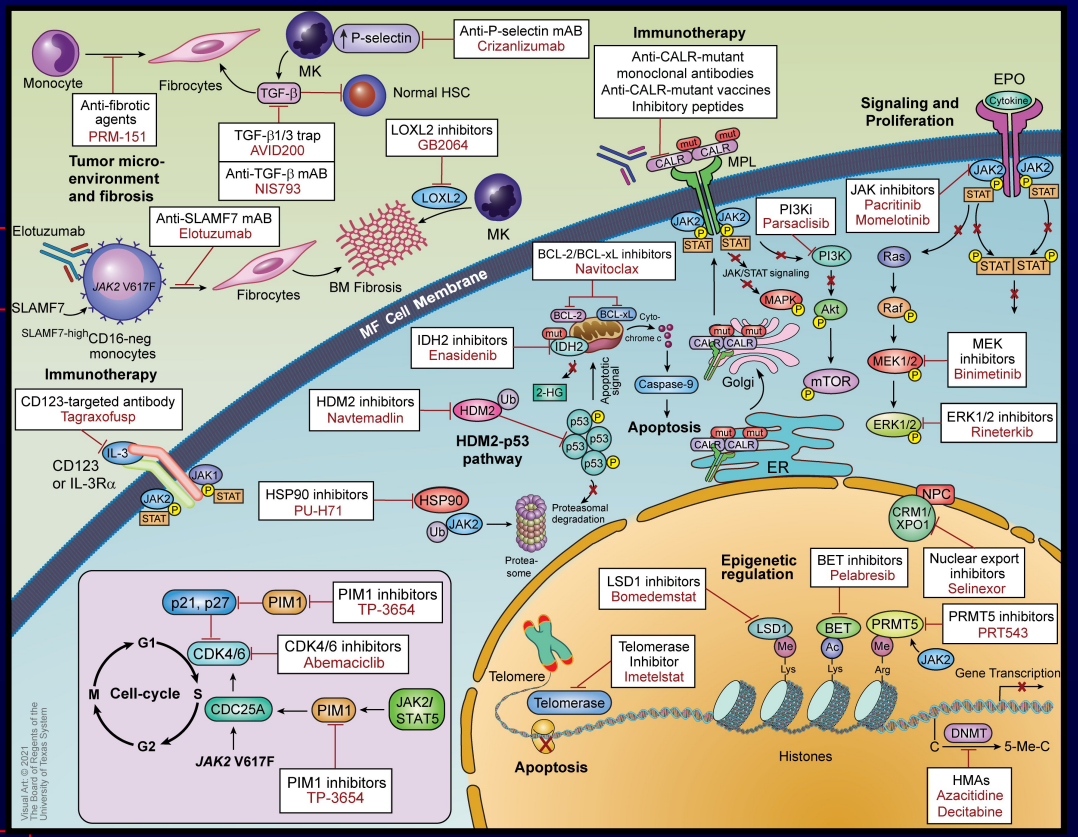
Targets of Novel Therapeutic Agents in Development for Myelofibrosis



Chifotides HT, Bose P, Masarova L. Pemmaraju N, Verstovsek S. *Clin.Lymph. Myeloma Leuk.* 2022; 22(4):210-223.

Ph I/II Combinations: JAKi + another agent/ “add-back or add-on” for MF

- RUXO + AZA – frontline (MF) and MDS/MPN-U
  - MDACC : MF (Masarova et al BLOOD 2018)
- RUXO + HSP90i (MF)
  - ClinicalTrials.gov Identifier: NCT03373877
- RUXO + BCL-xLi (MF) (Navitoclax)
  - ClinicalTrials.gov Identifier: NCT03222609
- RUXO + PI3Ki (MF) (Parsaclisib)
  - ClinicalTrials.gov Identifier: NCT01730248
- RUXO + THAL (MF) - frontline & R/R
  - ClinicalTrials.gov Identifier: NCT03069326
- RUXO + HDACi (Pracinostat) (MF) – frontline
- RUXO + IFN (2 ongoing clinical trials – Europe)
- RUXO + BETi (MF) (Pelabresib)
- RUXO + Sotatercept /Luspatercept
- RUXO + MDM2i (Navtemadlin)
- RUXO + XPO1i (Selinexor)



Economides M, Verstovsek S, Pemmaraju N Curr Hematol Malig Rep. 2019 Aug 1

Allo SCT in MF=only curative approach in MF

- **Leukemia, 2015: Consensus working group:**
  - Int-2 or high risk MF & age <70 should be considered
  - Int-1 and age <65 should be considered if either refractory, tx-dep anemia or a % of blasts in periph blood >2%, or unfav cytogen
- **Popat et al : ASCO 2015:**
  - Final prospective ph 2 results: flu/bu conditioning pts with MF
  - n-=46; 50% male, med age 58 [27-74 y]
  - Int risk (28) or high risk (18)
  - All pts engrafted; median time 13 days (neutrophil)
  - Med f/u of 5.1 years (1-8.3 y); 3 yOS: 69%; 3yEFS: 48%; CI relapse: 39%

- Questions remaining: JAKi pre/post; splenectomy pre-/post, timing of SCT, pt populations

MPN Clinical Pearls for : MF

- Include thinking about MPNs /MF in workup of patient with unknown hepatosplenomegaly, including in the young patient
- Pay attention to CBC and especially diff: look for circulating blasts, immature granulocytes, metamyelocytes, “tear drop” cells
- Remember: MF can transform to AML, early death rates; allo-SCT is potentially curative for intermediate/higher risk; remember referral for clinical trials including frontline

Thank you: To our Rapidly Growing MPN Community

- Please email me npemmaraju@mdanderson.org or call me 713-792-4956 if you have any questions

- #MPNSM: Twitter @doctorpemm

- Ruben Mesa
- Marina Konopleva
- Hagop Kantarjian
- Alison Moliterno
- Nitin Jain
- Tapan Kadia
- Naval Daver
- Gautam Borthakur
- Serge Verstovsek
- Kapil Bhalla
- Jeff Tyner
- Jerry Spivak
- Prof. Claire Harrison
- Richard Silver
- John Mascarenhas
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- Jean-Jacques Kiladjian
- Vikas Gupta
- Mike Thompson
- Jason Gotlib
- Raajit Rampal
- Rami Komrokji
- Nicole Kucine
- P Bose, L Masarova, S Pierce, Lisa Zhou
- M Patnaik, A Yacoub
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- MD Anderson Cancer Network
- Rhona Pinsoy & MPN Research/Protocol team
- Carol Bivins, Silvia Rivera
- Jyoti Nangalia
- Jeanne Palmer
- Steve Oh, Shannon Elf, Ami Patel, Gaby Hobbs
- Deepti Radia
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