MPN Therapy in 2021

18th Indy HEME Review

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Mays Cancer Center
UT Health San Antonio MD Anderson Cancer Center

Disclosures

Consulting: Novartis, La Jolla, Samus, Sierra Oncology, Blueprint, Abbvie, BMS, Genentech, Roche, Geron

Research Support: Incyte, Celgene, CTI, Promedior, Genentech, Abbvie, Imago

Off Label Use: Hydroxyurea, PEG Interferon, ruxolitinib

Learning Objectives

1. Better understand the factors leading to the burden of having an MPN

2. Understand the role of cytoreduction for essential thrombocythemia

3. Understand evolving role of interferons for polycythemia vera and new upcoming agents

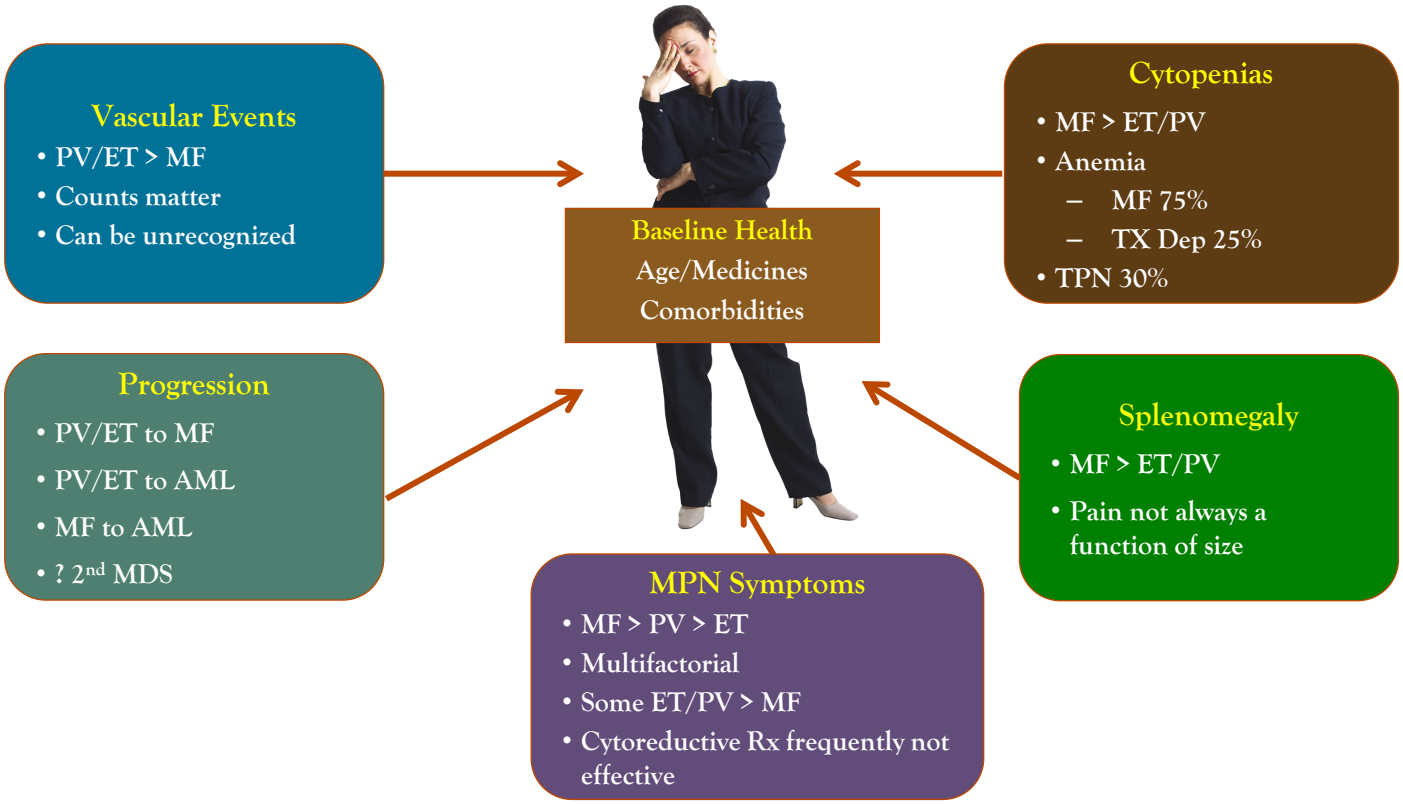
4. Understand evolving front and second line agents for myelofibrosis

MPNs 2021

- Burden of Having an MPN
- Essential Thrombocythemia
- Polycythemia Vera
- Myelofibrosis
- Complementary Approaches

Assessing MPN Burden

WHO Diagnosis Does Not Tell Whole Story



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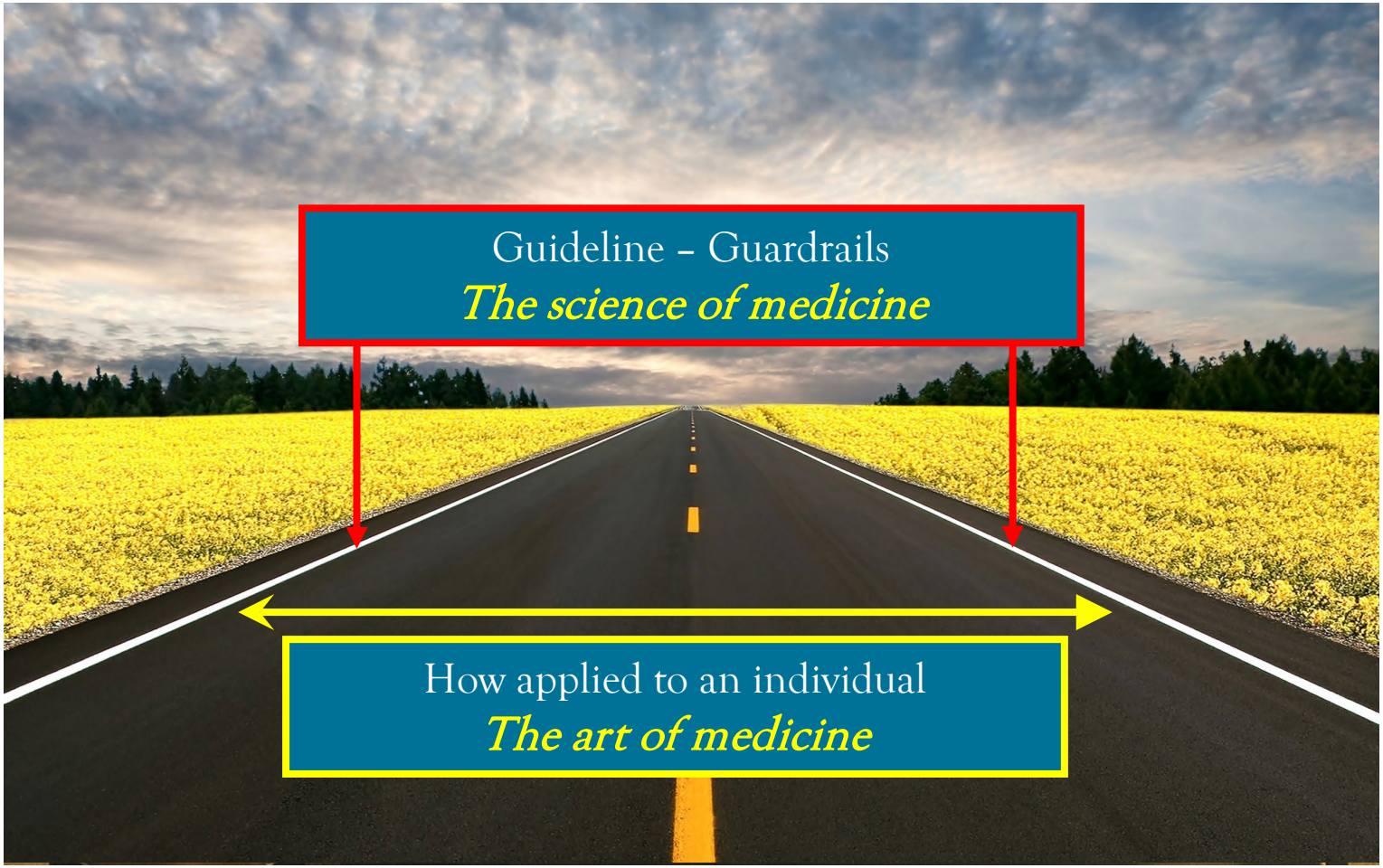
Treatment Goals

- Avoiding thrombosis and bleeding?
- Improving MPN associated symptoms?
- Increase activity?
- Decreasing splenomegaly?
- Improving anemia?
- Improving low platelets?
- Decreasing progression?
- Preventing progression?
- Live longer?

What is a treatment guideline?



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MPNs 2021

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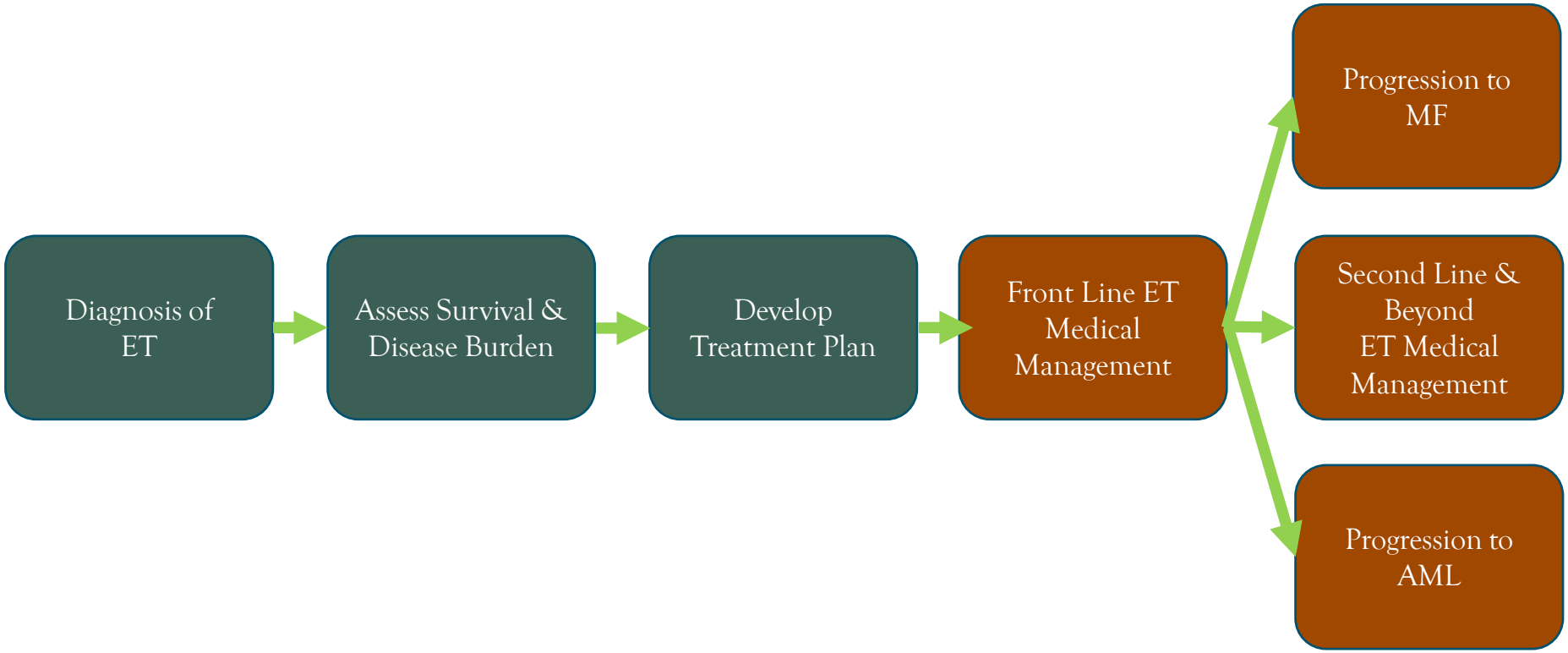
Management of ET 2021



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Pipeline – PV and ET

PV	ET
Hepcidin Agonists (PTG-300)	RoPEG vs ANAG
HDAC Inhibition (Givinostat)	IMG7289
MDM2 Inhibitors Idasanutlin-KRT232	
Other?	Other?

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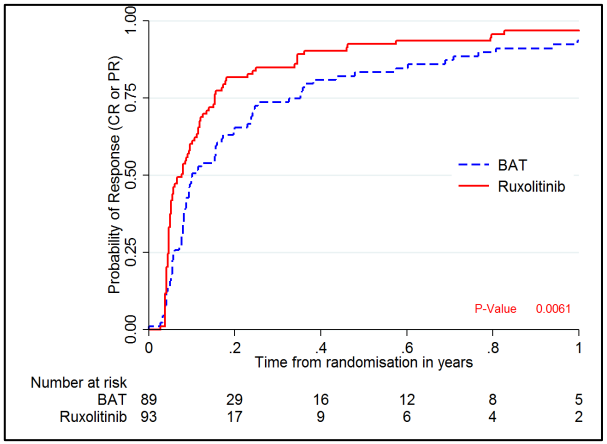
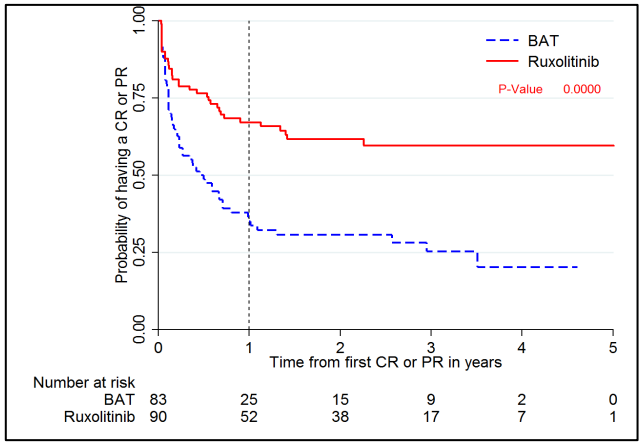
MAJIC PV: A randomised study of ruxlotinib vs BAT in HC resistant/intolerant PV (& ET)

Real world study from UK no cross-over

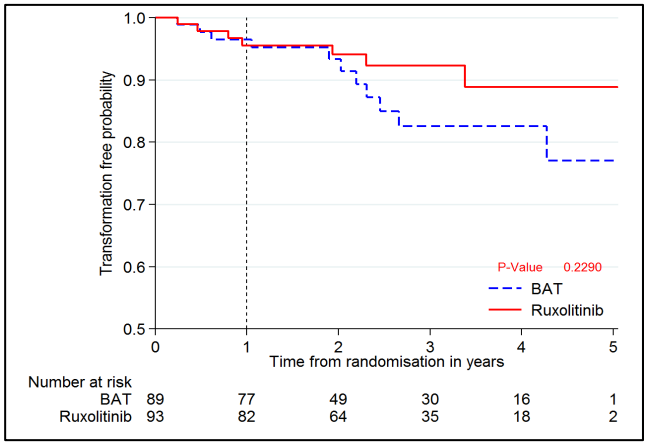
Speed of attaining response



Duration of overall response



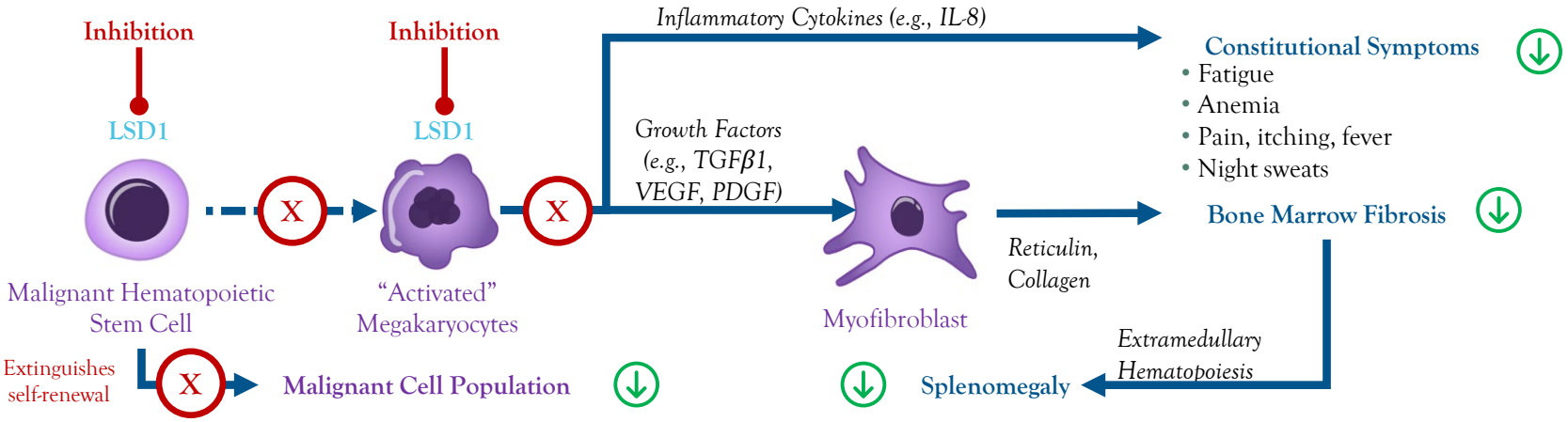
Transformation free survival



Harrison C, et al, PF625, EHA 2018

LSD1 Inhibition has Strong Therapeutic Rationale in MPNs

- LSD1 inhibition impairs function of both activated megakaryocytes and malignant stem cells
- Megakaryocytes produce cytokines and growth factors that drive myelofibrosis



LSD1 inhibition reduces production of megakaryocytes, growth factors and cytokines = symptom improvement

Potential to extinguish self-renewal of malignant stem cells = potential to improve overall survival

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CTP-201 Study Design in ET

Essential Thrombocythemia (ET)

- Rare chronic blood disorder driven by sustained overproduction of platelets by megakaryocytes in the bone marrow
- Characterized by microcirculatory symptoms and risk of thrombosis, bleeding and related cardiovascular events and can progress to AML
- 80k high-risk patients eligible for bomedemstat in US
- Bomedemstat safely reduces platelets in MPNs

Phase 2b Protocol Synopsis

- FDA approved in 2019
- Up to 60 high-risk ET patients
- Open label, bomedemstat once-daily
- ~25 sites in US, UK, EU, AUS and NZ
- **Primary objectives**:
  - Safety and tolerability
  - Reduction of platelet count to ≤400k/µL
- **Secondary objectives**
  - Reduction in VAF
  - Reduced event rate (thrombosis and hemorrhage)
  - Reduced progression to MF and AML

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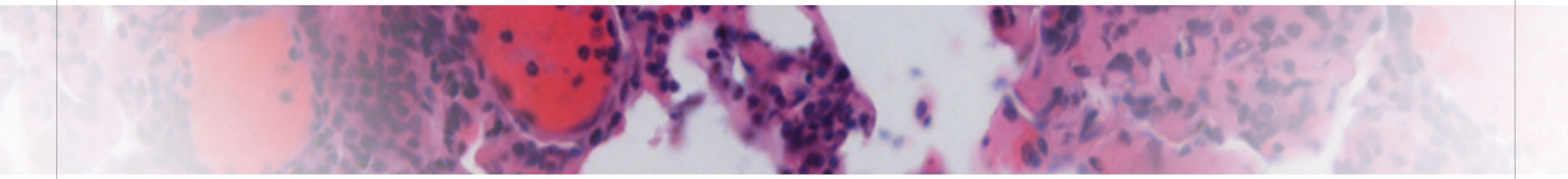
American Society *of* Hematology

Helping hematologists conquer blood diseases worldwide



Symptom Burden and Quality of Life in High-Risk ET and PV Patients Receiving Hydroxyurea or Pegylated Interferon Alfa-2a: Results of MPN-RC 111 and 112 Trials

Gina L. Mazza

on behalf of

Carolyn Mead-Harvey, John Mascarenhas, Abdulraheem Yacoub, Ronald Hoffman, Heidi E. Kosiorek, Josef T. Prchal, Richard T. Silver, Tiziano Barbui, Amylou C. Dueck, Ruben A. Mesa

MAYO CLINIC

Results – Patients

Characteristic	MPN-RC 111		MPN-RC 112	
	ET (*n* = 64)	PV (*n* = 50)	ET (*n* = 79)	PV (*n* = 87)
Sex (% Female)	51%	48%	50%	33%
Age in Years (Median, Range)	65 (20 – 85)	64 (26 – 84)	60 (18 – 83)	62 (20 – 88)
Months Since Dx (Median, Range)	38 (0 – 291)	55 (1 – 394)	3 (0 – 48)	3 (0 – 84)
Prior Thrombosis (%)	31%	22%	25%	29%
Splenomegaly (%)	19%	56%	11%	37%

Results – Symptoms

- MPN-RC 111 patients had significant **improvement** of TSS, fatigue, abdominal pain, abdominal discomfort, dizziness, numbness, night sweats, and fever
- MPN-RC 112 PEG patients had significant **worsening** of fever
- MPN-RC 112 HU patients had significant **worsening** of inactivity
- MPN-RC 111 and 112 PEG patients had significant **worsening** of PEG-related symptoms

61st Annual Meeting & Exposition of the American Society of Hematology

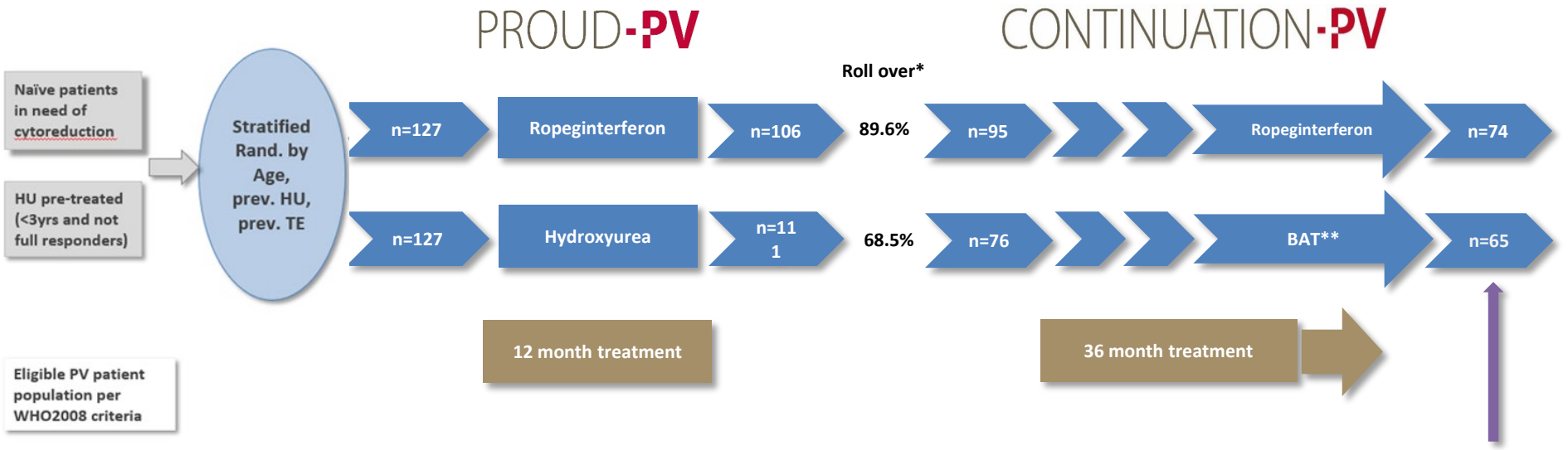
THROMBOEMBOLIC RISK REDUCTION AND HIGH RATE OF COMPLETE MOLECULAR RESPONSE WITH LONG-TERM USE OF ROPEGINTERFERON ALPHA-2B IN POLYCYTHEMIA VERA: RESULTS FROM A RANDOMIZED CONTROLLED STUDY

Jean-Jacques Kiladjian,

Christoph Klade, Pencho Georgiev, Dorota Krochmalczyk, Liana Gercheva-Kyuchukova, Miklos Egyed, Viktor Rossiev, Petr Dulicek, Arpad Illes, Halyna Pylypenko, Lylia Sivcheva, Jiri Mayer, Vera Yablokova, Kurt Krejcy, Hans C. Hasselbalch, Robert Kralovics and Heinz Gisslinger

for the PROUD-PV Study Group

Phase III: PROUD- and CONTINUATION-PV



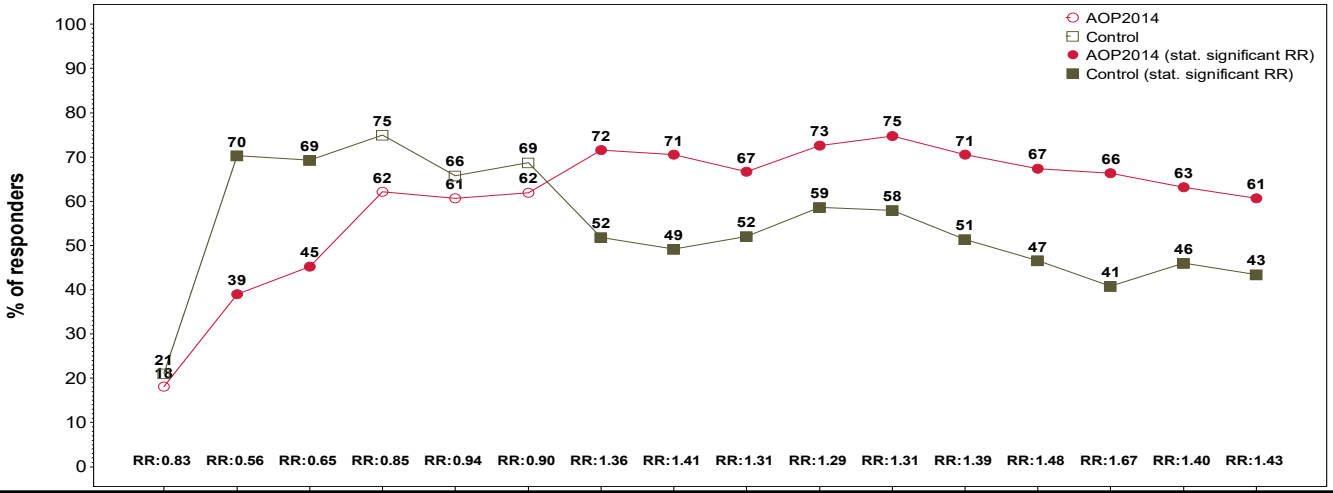
* There were no significant differences between patients who entered the extension study and those who did not roll-over.

**Control group received best available treatment (BAT; 97% of patients received hydroxyurea as of last available assessment)

Data snapshot:

- Efficacy data up to 48 months
- All safety data (up to 64 months)

Complete Hematologic Response (CHR)



Study Month	Responder/N	Responder %	Responder/N	Responder %	P-value	RR [95% CI] (AOP2014/Control)
	Ropeginterferon (N=95)		Control (N=76)			
Month 12 (End of PROUD-PV)	59/95	62.1	57/76	75.0	0.1211	0.85 [0.70-1.04]
Month 24	67/95	70.5	33/67	49.3	0.0117	1.41 [1.08-1.85]
Month 36	67/95	70.5	38/74	51.4	0.0108	1.39 [1.08-1.79]
Month 48	57/94	**60.6**	33/76	**43.4**	**0.0194**	**1.43 [1.06–1.93]**

Major Thromboembolic Events (Derived from Safety Data)

- Observational period: 419 patients-years in Ropeginterferon arm, 338 patient-years in control arm

Baseline characteristics								
Patients	**Sex**	**Prior HU**	**PV duration (months)**	**Age (years)**	**Prior TE events**	**Thromboembolic event**	**Onset (days since first dose)**	**CHR prior to event**
Ropeginterferon								
1	M	No	0.8	61	No	**Haemorrhagic transformation stroke** **Ischemic stroke**	63 1406	Not available Yes
2	F	Yes	1.7	61	No	**Ischemic stroke**	95	No
3	M	No	0.5	64	Yes	**Splenic infarction / truncus coeliacus thrombosis**	183	No
4	M	Yes	26.0	67	No	**Intracardiac thrombus**	280	No
Control								
1	M	No	0.6	77	No	**Femoral artery occlusion**	131	No
2	F	No	7.6	67	No	**Embolism**	176	Yes
3	F	No	17.0	65	Yes	**Thrombophlebitis superficial**	713	No
4	F	No	0.1	76	No	**Venous thrombosis limb**	1016	No

Malignancies

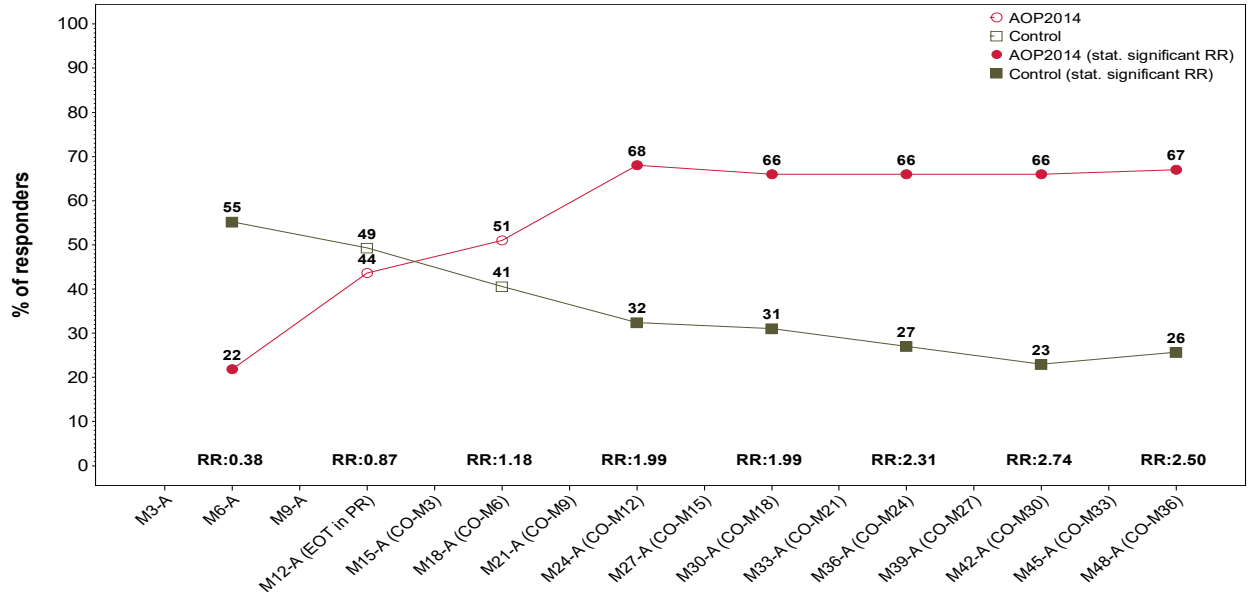
Year of treatment	Treatment- or PV-related neoplasms (investigator's assessment)	
	Ropeginterferon	**Control**
1	-	Basal cell carcinoma (2 cases)
2	-	Acute leukemia (2 cases) Malignant melanoma
3	-	-
4	-	Myelofibrosis
> 4	Myelofibrosis	-
Total	**1**	**6**

All neoplasms, including those unrelated to PV or treatment:

Ropeginterferon arm: 16 cases in 12 patients

Control arm: 15 cases in 12 patients

Molecular Response



Study Month	Responder/N	Responder %	Responder/N	Responder %	P-value	RR [95% CI] (AOP2014/Control)
	Ropeginterferon (N=95)		**Control (N=76)**			
Month 12 (End of PROUD)	41/94	43.6	36/73	49.3	0.3706	0.87 [0.63-1.19]
Month 24	64/94	68.1	24/74	32.4	<0.0001	1.99 [1.41-2.82]
Month 36	62/94	66.0	20/74	27.0	<0.0001	2.38 [1.56-3.42]
Month 48	63/4	**67.0**	19/74	**25.7**	**<0.0001**	**2.50 [1.68-3.72]**

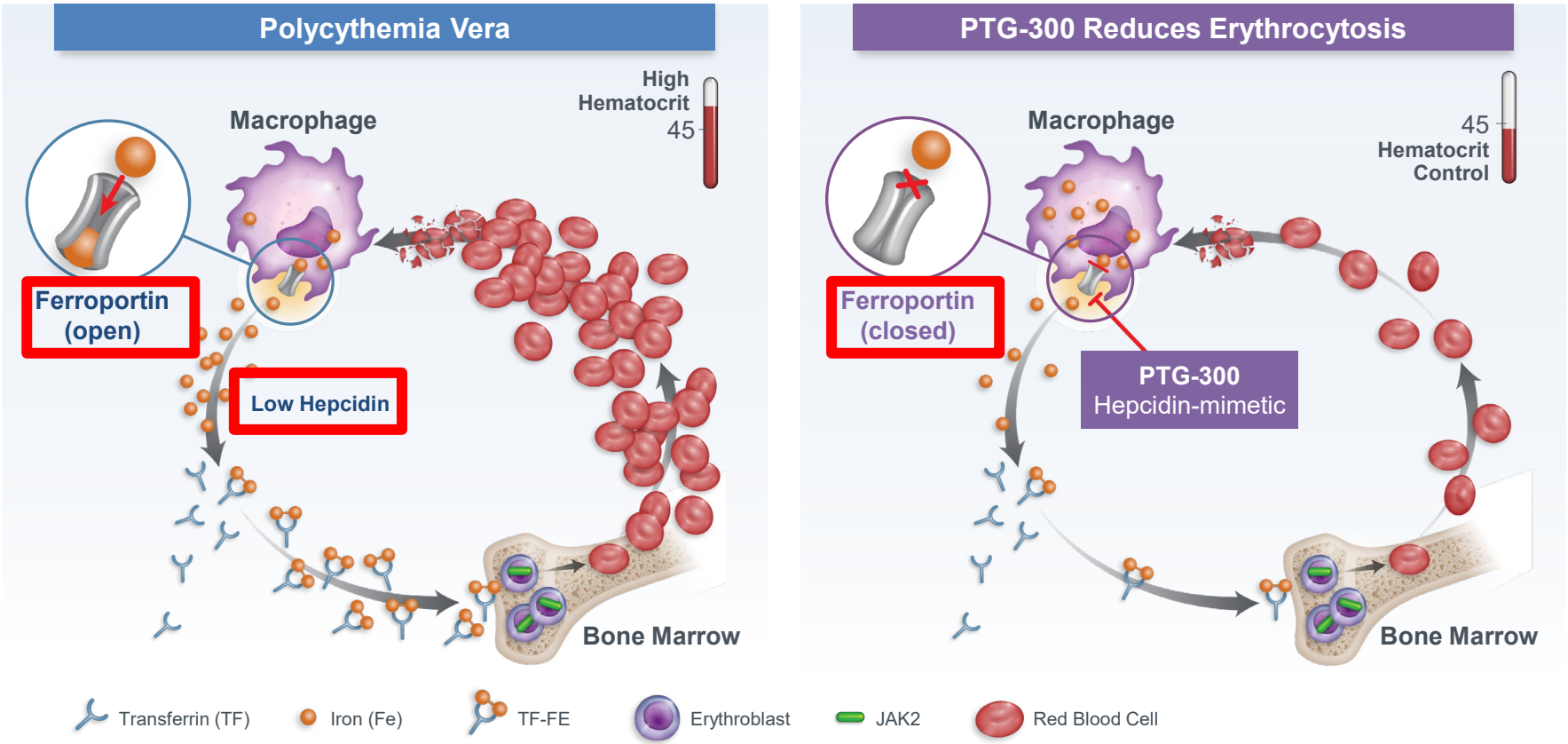
PTG-300 Eliminates the Need for Therapeutic Phlebotomy and Reverses Iron Deficiency in Both Low and High-risk Polycythemia Vera Patients

Marina Kremyanskaya[1], Yelena Ginzburg[1], Andrew Kuykendall[2], Naveen Pemmaraju[3], Abdulraheem Yacoub[4], Jay Yang[5] , Frank Valone[6], Sarita Khanna[6], Suneel Gupta[6], Srdan Verstovsek[3], Ronald Hoffman[1]

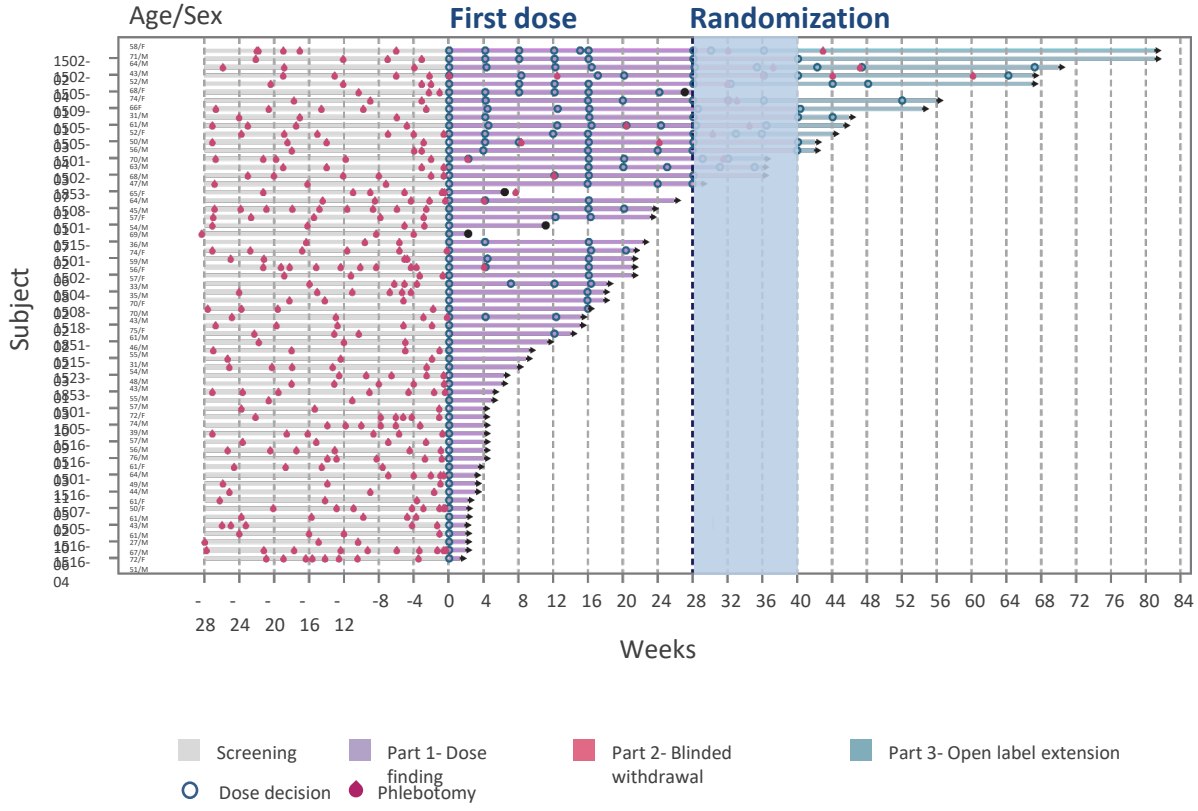
[1]Hematology and Medical Oncology, Mount Sinai Hospital, New York, [2]Malignant Hematology , Lee Moffitt Cancer Center , Tampa, FL, [3]Dept of Leukemia, MD Anderson, Houston, [4]Cancer Center, University of Kansas Cancer Center, Westwood, KS, [5]Hematology, Karmanos Cancer Institute, Detroit, [6]Clinical development, Protagonist Therapeutics, Newark, United States

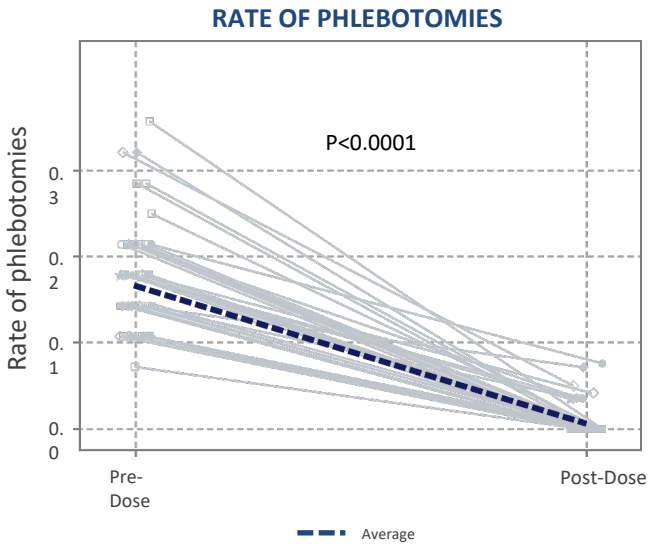
Program Section: Novel Therapies and Targets in MPN

PTG-300 (Rusfertide) Mechanism of Action



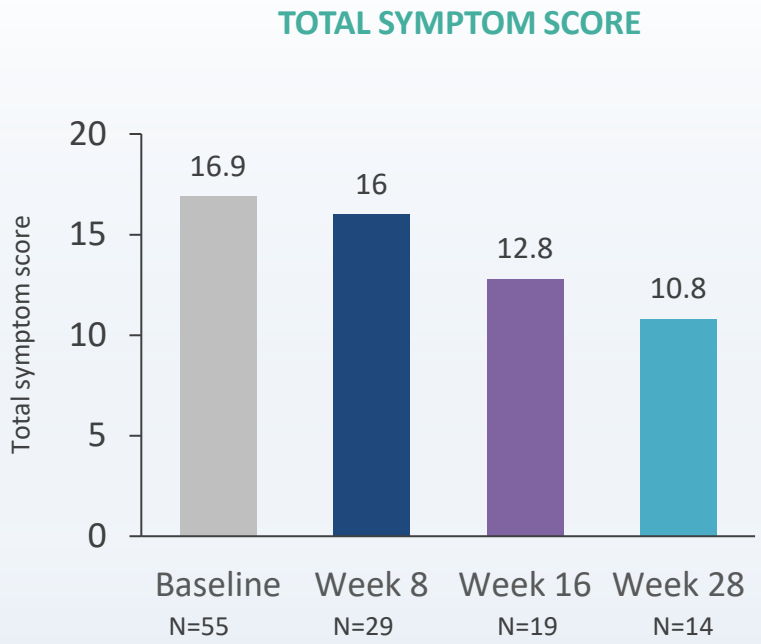
Therapeutic Phlebotomies Prior to and on PTG-300

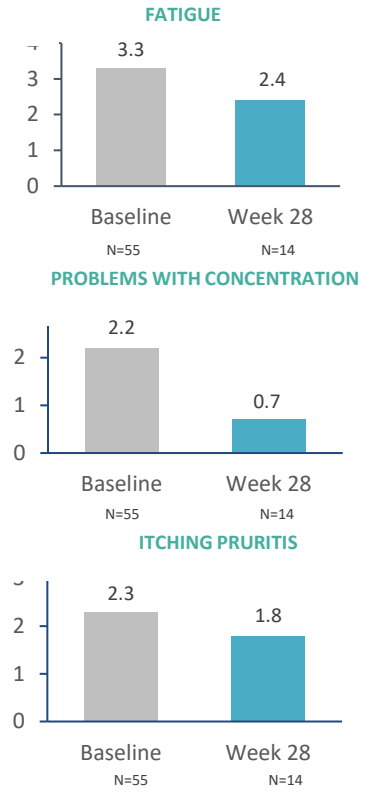




Kremyanskaya EHA 2021

Improvement in MPN-TSS Scores Following PTG-300





Kremyanskaya EHA 2021

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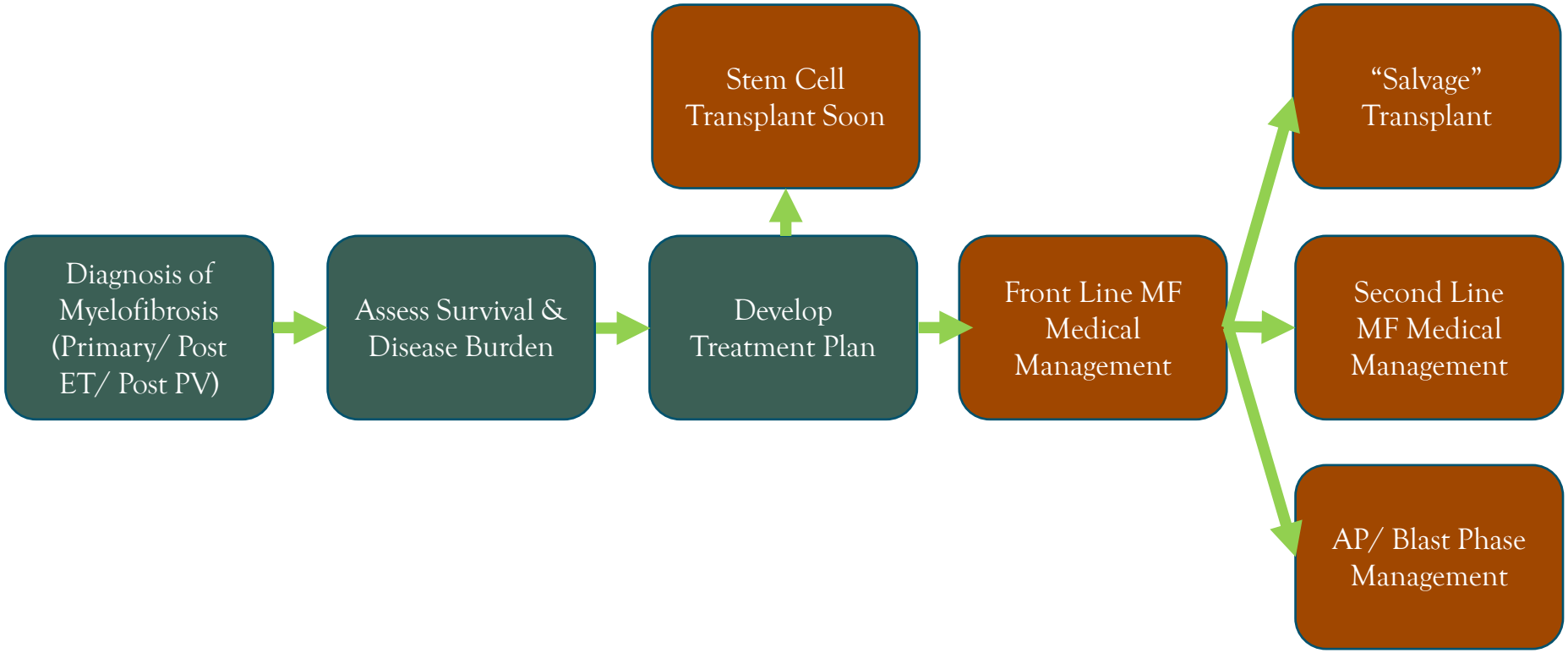
Management of Myelofibrosis 2021



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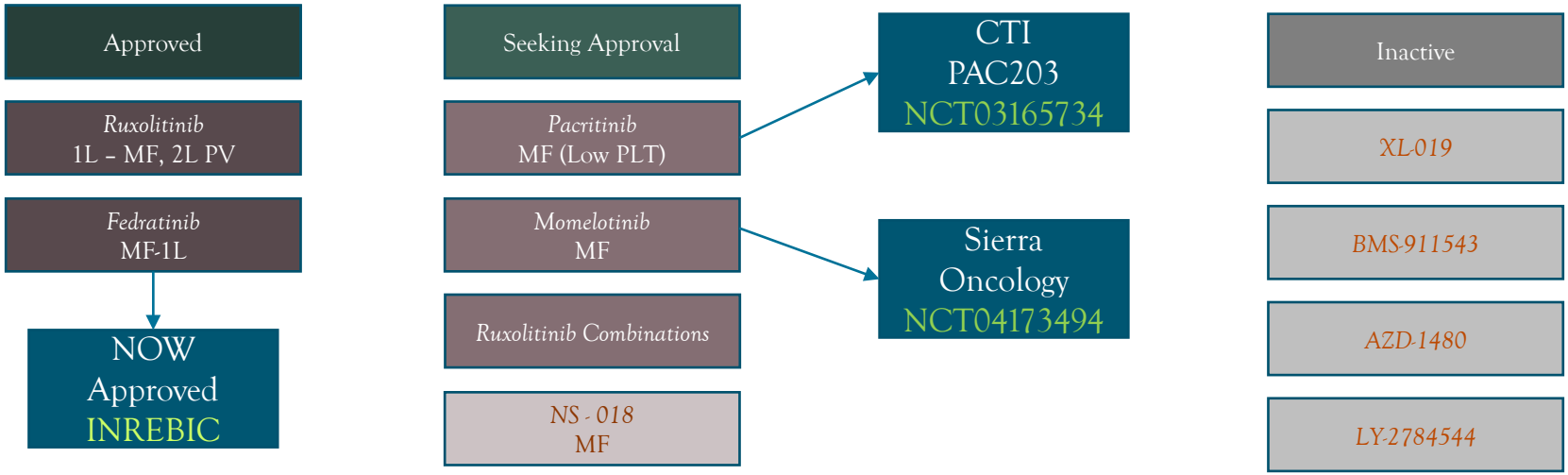
JAK Inhibitor Landscape 2021



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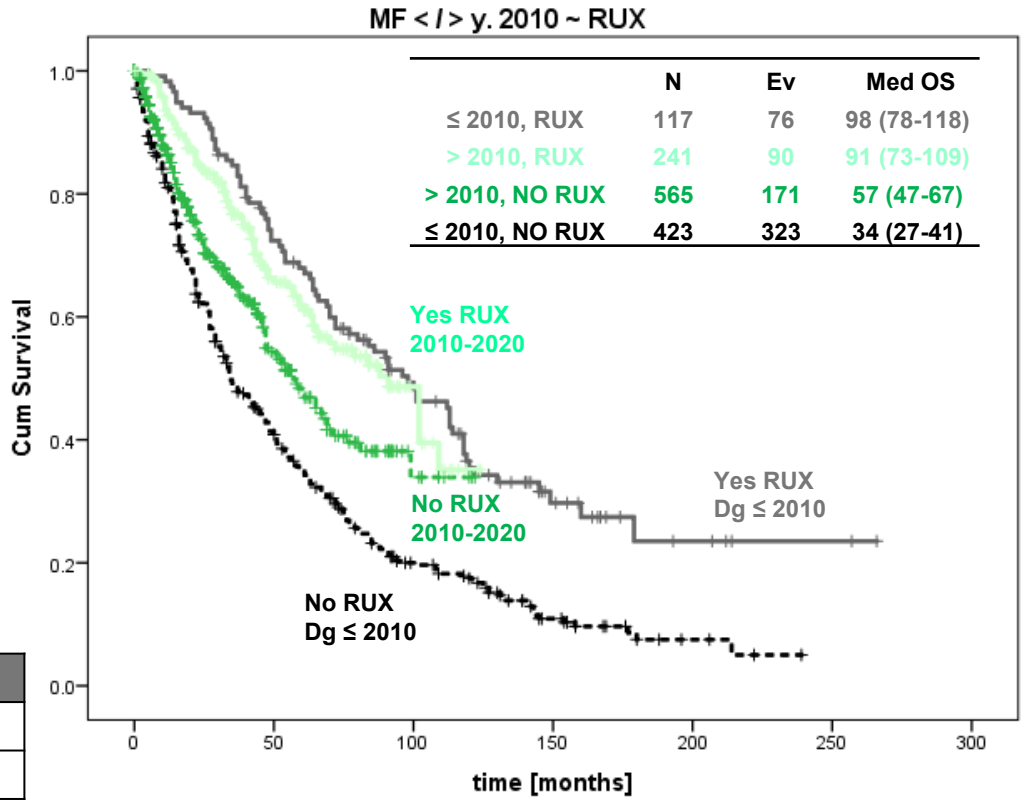
Takeaway 1:

Effective MF Therapies May Prolong Overall Survival

Results: OS ~ Ruxolitinib



OS without and with rux ~ years 2010
≤ y. 2010, p < 0.001, HR 0.46, 95% CI 0.35-0.59
> y. 2010, p = 0.001, HR 0.66, 95% CI 0.51-0.85

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
Making Cancer History®

Masarova et. al. ASH 2020

EUROPEAN HEMATOLOGY ASSOCIATION

Overall and progression-free survival in patients treated with fedratinib as first-line myelofibrosis therapy and after prior ruxolitinib: results from the JAKARTA and JAKARTA2 trials

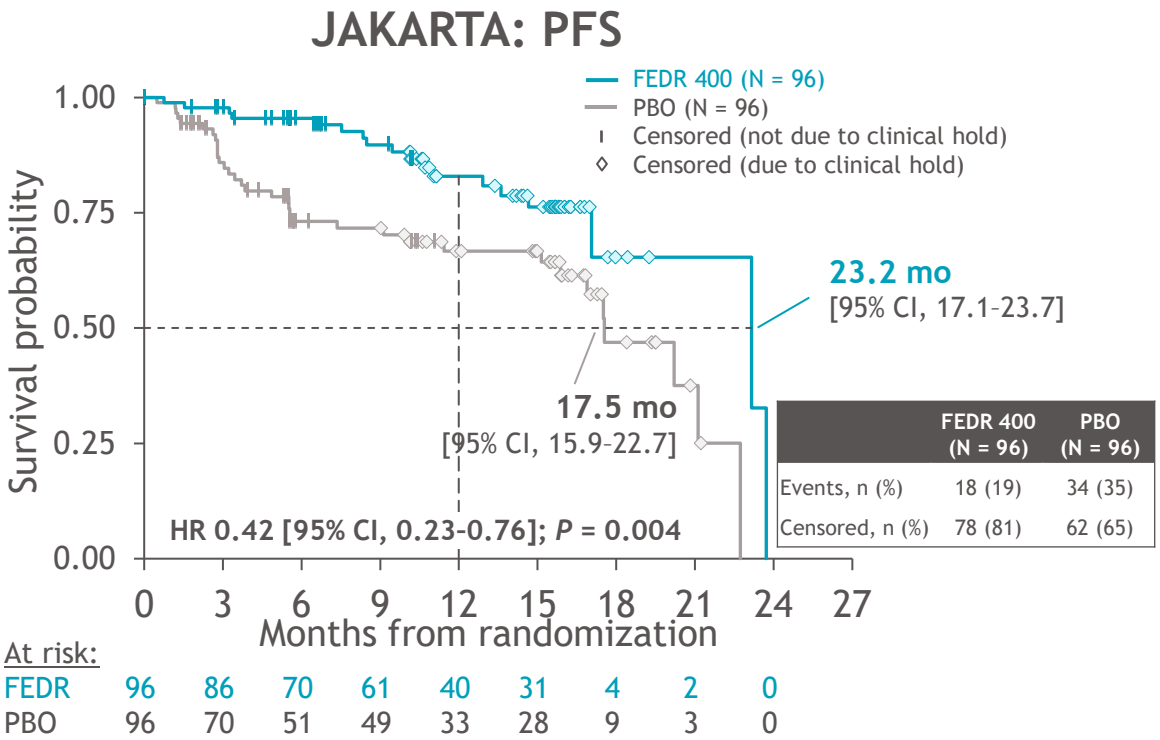
Claire Harrison,[1] Jean-Jacques Kiladjian,[2] Srdan Verstovsek,[3] Alessandro Vannucchi,[4] Ruben Mesa,[5] Andreas Reiter,[6] Jun Zhang,[7] Shelonitda Rose,[7] and John Mascarenhas[8]

[1]Guy's and St Thomas' Hospital, London, United Kingdom; [2]Hôpital Saint-Louis and Université de Paris, Paris, France; [3]The University of Texas MD Anderson Cancer Center, Houston, United States; [4]AOU Careggi, University of Florence, Florence, Italy; [5]Mays Cancer Center at UT Health San Antonio MD Anderson, San Antonio, United States; [6]Universitätsklinikum Mannheim, Mannheim, Germany; [7]Bristol Myers Squibb, Princeton, United States; [8]Icahn School of Medicine at Mount Sinai, New York, United States

Presentation S203

JAKARTA: Progression-free survival

- **FEDR 400 significantly reduced the risk of disease progression vs. PBO (*P* = 0.004)**
  - Median PFS was 5.7 months longer in the FEDR 400 arm vs. PBO: 23.2 vs. 17.5 mo, respectively
  - 1-year PFS: FEDR 400 83%, PBO 67%
- **80 pts (42%) were still being followed for PFS at the time of clinical hold**
  - Median follow-up: FEDR 400, 10.6 mo; PBO, 9.1 mo
- AML transformation was reported in 3 pts (3%) in the FEDR 400 arm and 2 pts (2%) in the PBO arm[a]

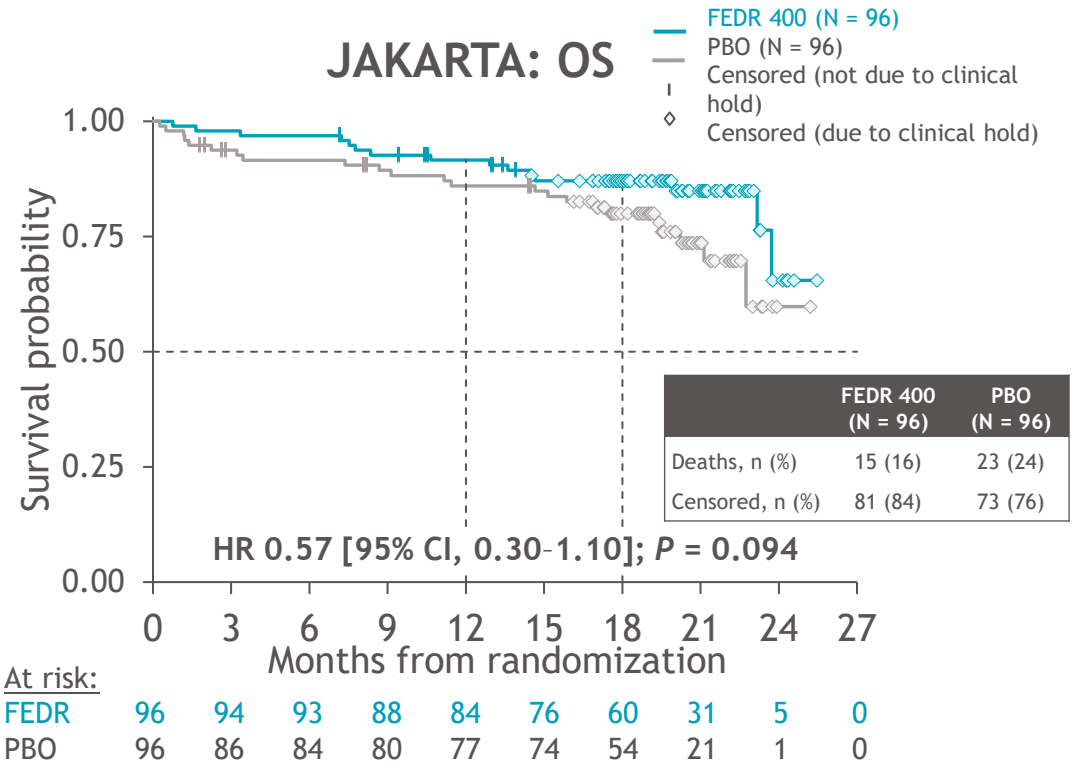


	FEDR 400 (N = 96)	PBO (N = 96)
Events, n (%)	18 (19)	34 (35)
Censored, n (%)	78 (81)	62 (65)

[a]AML transformation was based on adverse event reporting, including the preferred terms of "*Acute myeloid leukemia*", "*Acute leukemia*", and "*Transformation to acute myeloid leukemia*".
P value from log-rank test.
AML, acute myeloid leukemia; CI, confidence interval; HR, hazard ratio; FEDR, fedratinib; mo, months; PBO, placebo; PFS, progression-free survival; pts, patients.

JAKARTA: Overall survival (ITT)

- **Median OS was not reached (NR) in the FEDR 400 [95%CI, 23.7 mo - NR] or PBO [22.7 - NR] arm**
  - 1-year OS rates: FEDR 400 mg, 92%; PBO, 86%
  - 18-mo OS rates: FEDR 400 mg, 87%; PBO, 80%
- ITT analysis; 74% of PBO-randomized pts crossed-over to FEDR after EOC6
- **139 pts (72%) were censored for OS at the time of clinical hold**
  - Median follow-up: FEDR 400 mg, 19.3 mo; PBO, 18.8 mo



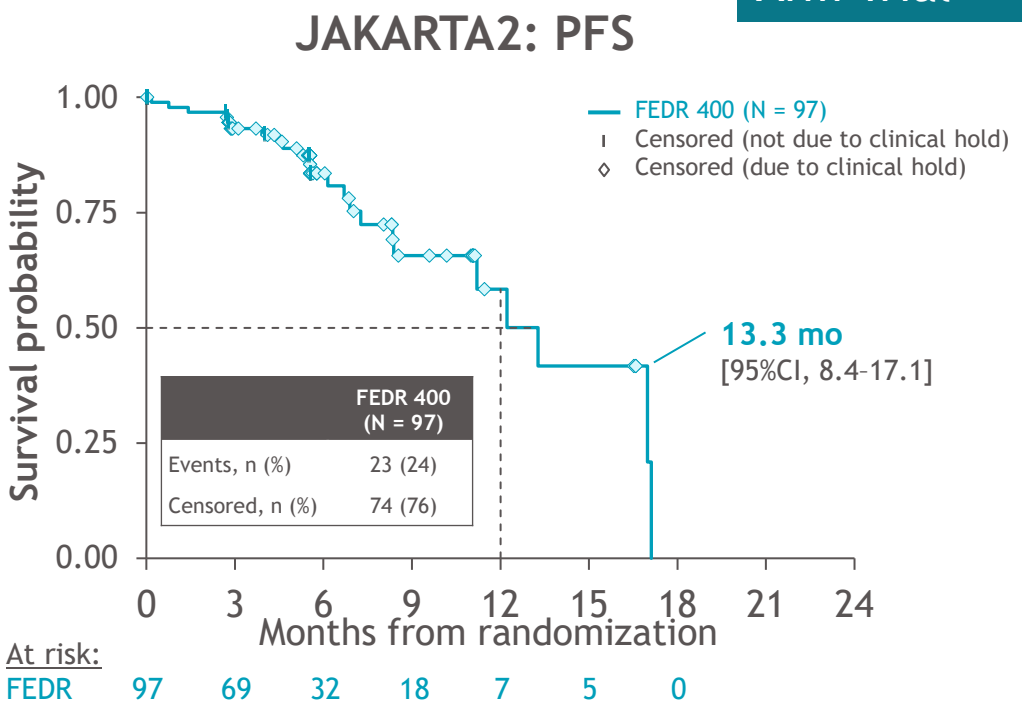
	FEDR 400 (N = 96)	PBO (N = 96)
Deaths, n (%)	15 (16)	23 (24)
Censored, n (%)	81 (84)	73 (76)

P value from log-rank test.
CI, confidence interval; HR, hazard ratio; FEDR, fedratinib; mo, months; NR, not reached; OS, overall survival; PBO, placebo; pts, patients.

JAKARTA2: Progression-free survival

Note: Single Arm Trial

- **Median PFS was 13.3 mo**
  - 1-year PFS rate was 59%
- **62 pts (64%) were still being followed for PFS at the time of clinical hold**
  - Median follow-up: 5.6 mo
- 2 pts (2%) experienced transformation to AML during the JAKARTA2 Tx period[a]

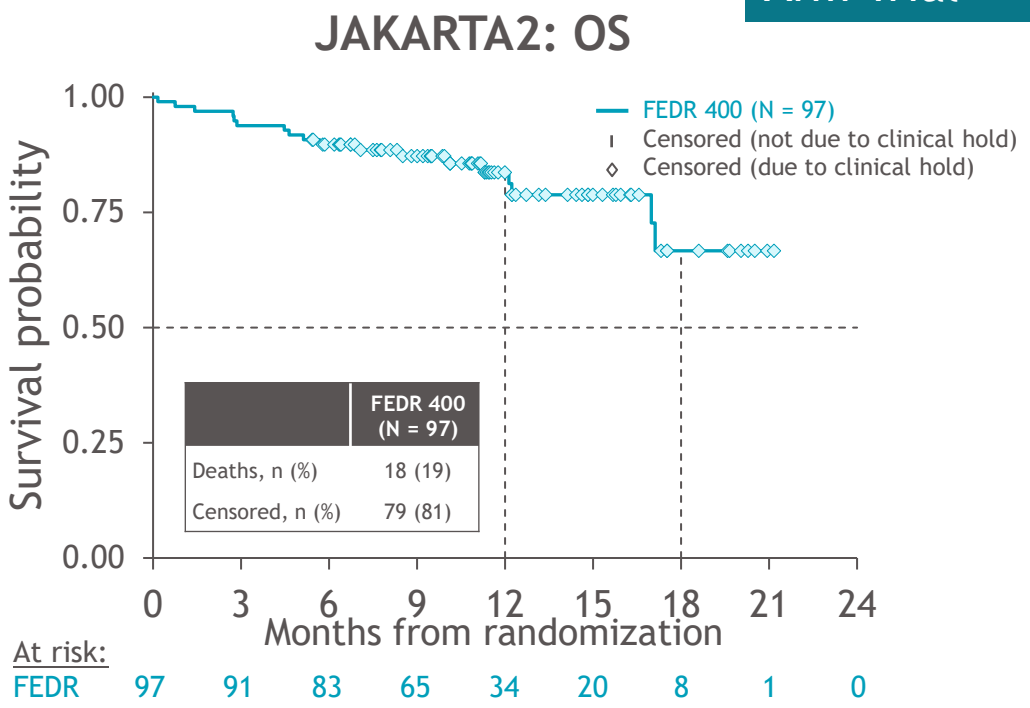


[a]AML transformation was based on adverse event reporting, including the preferred terms of "*Acute myeloid leukemia*", "*Acute leukemia*", and "*Transformation to acute myeloid leukemia*".
AML, acute myeloid leukemia; FEDR, fedratinib; mo, months; PFS, progression-free survival; pts, patients; Tx, treatment.

JAKARTA2: Overall survival

Note: Single Arm Trial

- **Median OS was NR [95%CI, 17.1 - NR]**
  - 1-year and 18-mo OS rates were 84% and 67%, respectively
- **79 pts (81%) were censored for OS at the time of clinical hold**
  - Median follow-up: 10.8 mo



FEDR, fedratinib; mo, months; NR, not reached; OS, overall survival; PBO, placebo; pts, patients.

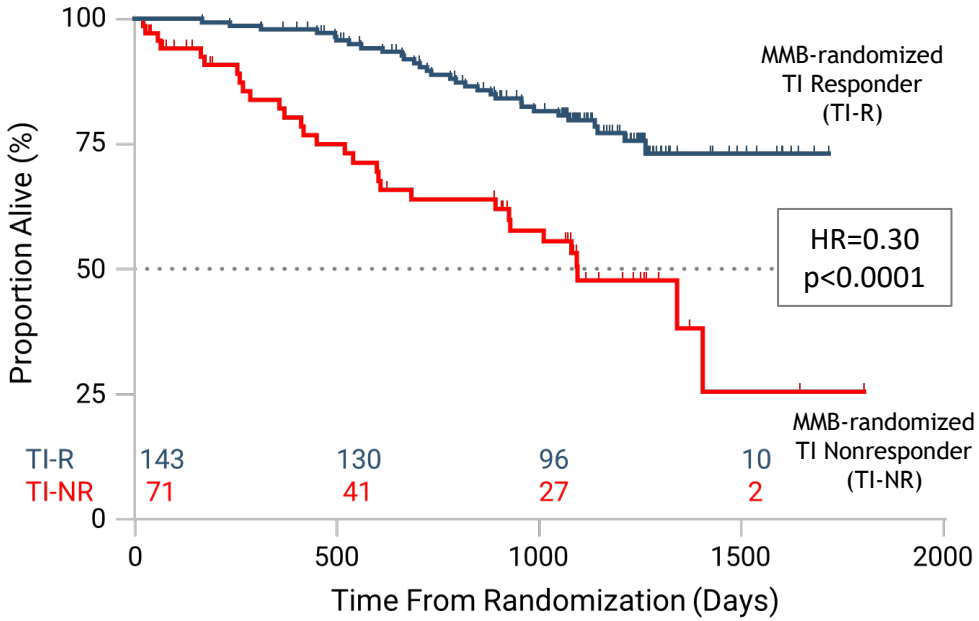
Transfusion Independence is Associated with Improved Overall Survival in Myelofibrosis Patients Receiving Momelotinib

Ruben Mesa*, Stephen T. Oh, Aaron T. Gerds, Vikas Gupta, John Catalano, Francisco Cervantes, Timothy Devos, Marek Hus, Jean-Jacques Kiladjian, Ewa Lech-Maranda, Donal McLornan, Jeanne Palmer, Uwe Platzbecker, Jacek Treliński, Kazuya Shimoda, Rafe Donahue, Bryan Strouse, Mark Kowalski, Srdan Verstovsek

*Mays Cancer Center, UT Health San Antonio, MD Anderson

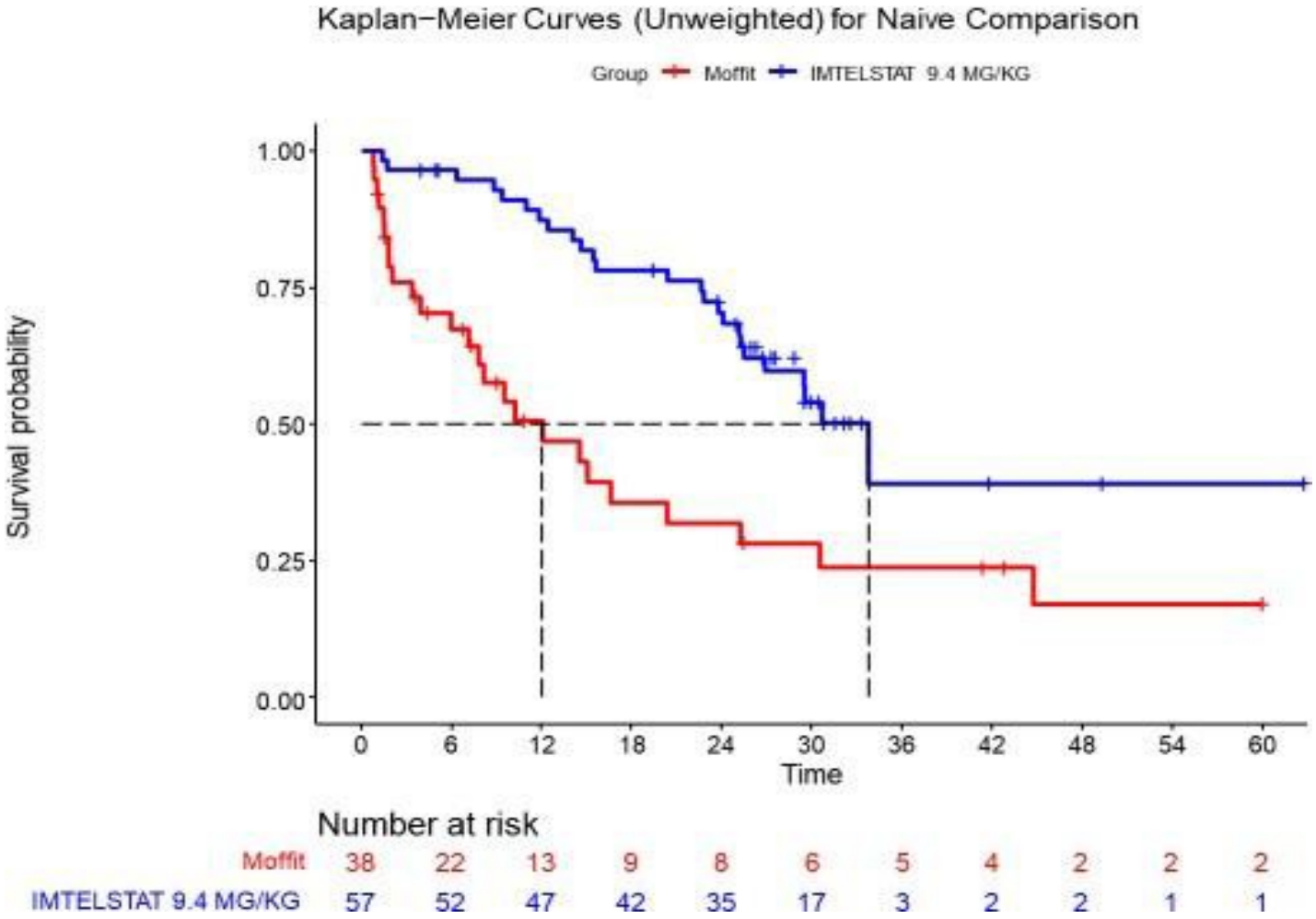
- Momelotinib (MMB) is a JAK1, JAK2 and ACVR1/ALK2 inhibitor
- Previously published data from the SIMPLIFY-1 Ph3 study of MMB vs ruxolitinib in JAKi-naïve patients show higher Week-24 (W24) transfusion independence (TI) responder rates on MMB (67%) vs RUX (49%)
- Correlation between W24 TI response and overall survival observed with MMB is unique and supports the clinical relevance of TI in patients with myelofibrosis receiving MMB: **3-year survival in MMB TI responders was 80%** compared to 50% in MMB TI nonresponders

SIMPLIFY-1 Patients Randomized to MMB Who Were TI Responders at W24 Show an OS Advantage



IMbark Phase 2 Imetelstat Data: Survival[1]

ClincialTrials.gov
(NCT02426086)



Median overall survival:
28.1 months (95% CI, 22.8 - 31.6)

After discontinuation of ruxolitinib[2-4]:
Median overall survival is ~14-16 months.

1. Mascarenhas J et al. EHA 2020. Abstract EP1107;
2. Kuykendall AT et al. *Ann Hematol.* 2018;97(3):435-441;
3. Newberry KJ et al. *Blood.* 2017;130(9):1125-1131;
4. Spiegel JY et al. *Blood Adv.* 2017;1(20):1729-1738.

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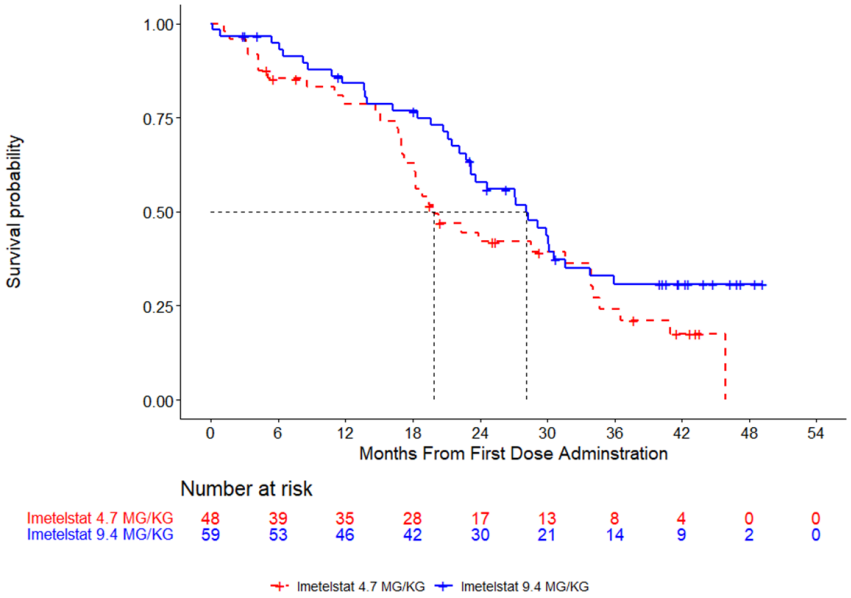
Potential OS Improvement with 9.4 mg/kg Imetelstat Treatment in Patients with MF R/R to JAKi



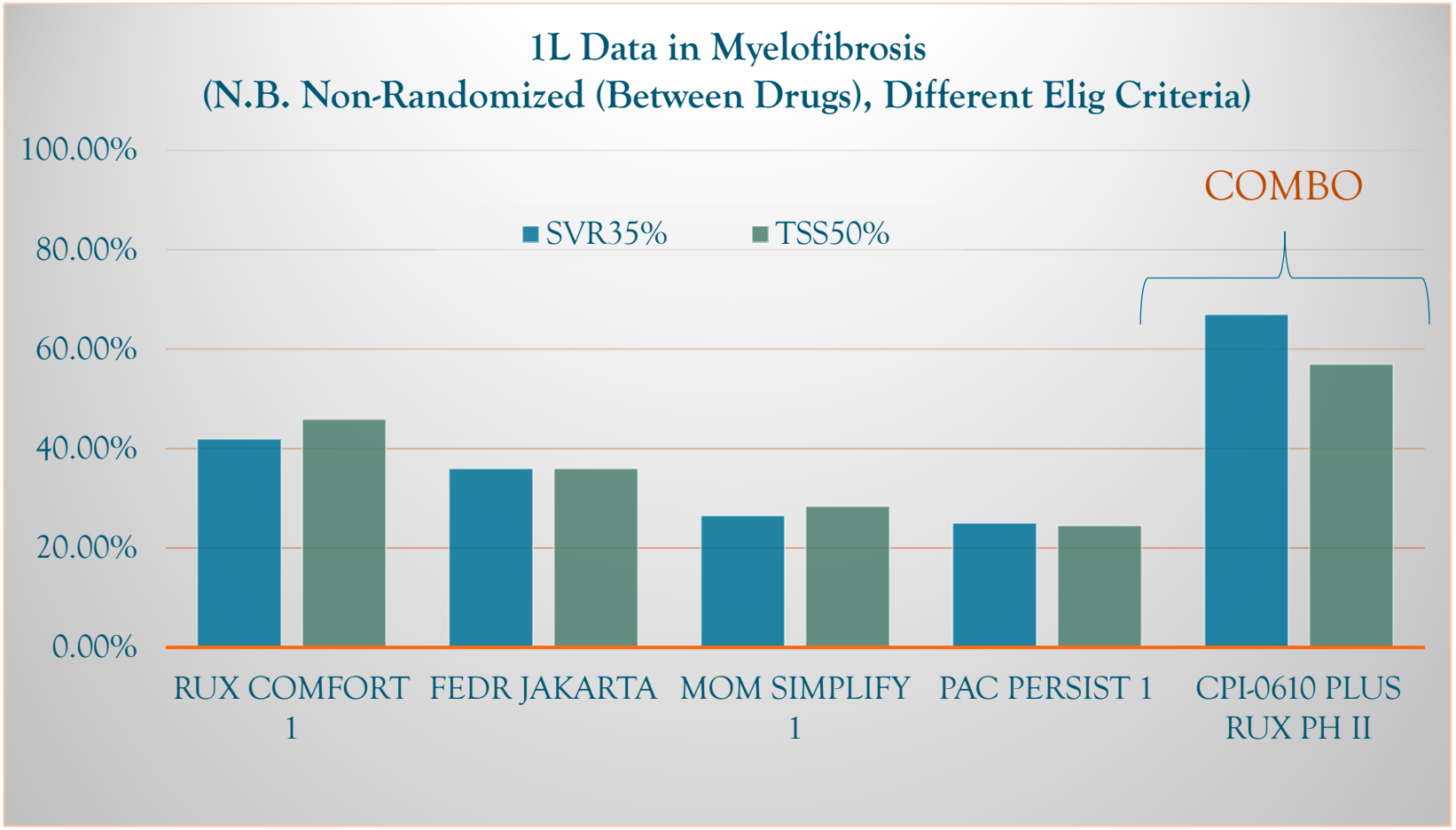
	4.7 mg/kg (N = 48)	9.4 mg/kg (N = 59)
Number of events, n (%)	35 (72.9%)	36 (61.0%)
Number censored, n (%)	13 (27.1%)	23 (39.0%)
Median Overall Survival (months) (95% CI)	19.9 (17.1, 33.9)	28.1 (22.8, 31.6)
12-months survival rate % (95% CI)	78.6 (63.9, 87.9)	84.0 (71.6, 91.4)
24-months survival rate % (95% CI)	42.0 (27.4, 56.0)	57.9 (43.6, 69.7)

OS analysis was performed based on database lock in April 2020; median follow-up was 41.7 months (range 0.2, 49.2)

Similar results were observed when sensitivity analyses accounted for confounding factors of subsequent therapies, including hematopoietic stem cell transplantation and dose escalation from 4.7 mg/kg to 9.4 mg/kg

Mascarenhas et. al. ASH 2020

Comparison for 1L MF Therapy



Verstovsek et. al. NEJM 2012
Pardanani et. al. JAMA Inc 2015
Mesa et. al. JCO 2017
Mesa et. al. Lancet Hematology 2017
Mascarenhas et. al. ASH 2020

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A selection of novel agents/targets being developed in MPN particularly MF

Cell-cycle Checkpoint

- P2 Imetelstat | Telomerase Inhibitor (*Geron*)
- P1 Alisertib | Aurora Kinase Inhibitor (*Takeda*)

Anti-fibrotic

- P2 PRM-151 | Pentraxin-2 (*Promedior*)

Receptor Ab / ADC

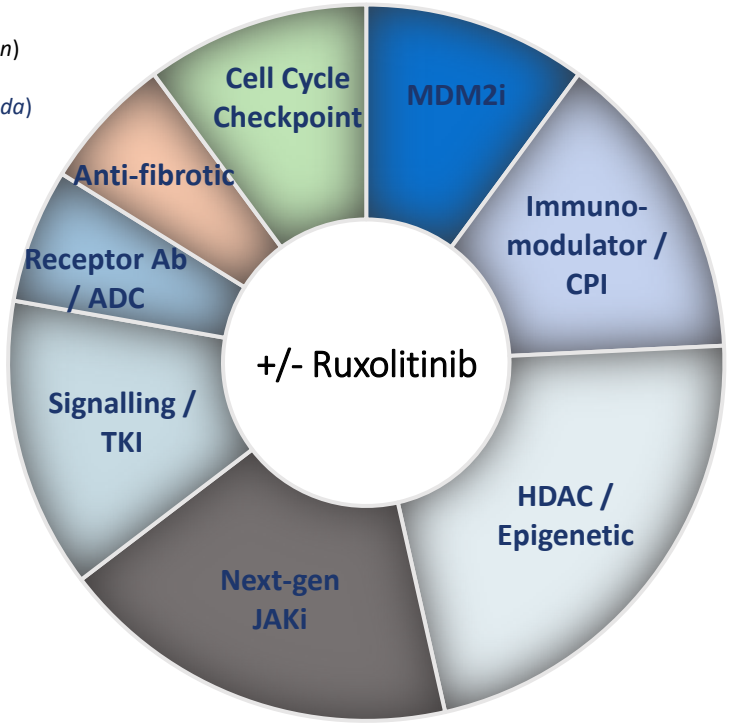
- P2 SL-401 | CD123-toxin (*Stemline*)

Signaling / TKI

- P2 Glasdegib | Hedgehog (*Pfizer*)
- P2 Sonidegib | Hedgehog (*Sun*)
- P2 INCB'465 | PI3Ki (*Incyte*)
- P2 LCL1 I SMAC/IAP (Novartis)

Next-gen JAKi

- P3 Fedratinib | JAK2 (*Celgene*)
- P3 Pacritinib | JAK2/FLT3 (*CTI Bio*)
- P3 Momelotinib | JAK2/1/ACVR1 (*Sierra*)
- P2 Itacitinib | JAK1 (*Incyte*)



Slide Courtesy of Prof Claire Harrison

Apoptosis/MDM2/BCL

- P1 KRT-232 (*Kartos Therapeutics*)
- P2 Idasanutlin / RG7388 (*Roche*)
- P1 Navitoclax | BCL2 inhibition (*Abbvie*)

Immuno-modulator / CPI

- P3 Pegasys | IFN-α2a (ESR/Roche)
- P3 Ropeg-IFN-α2b (*PharmaEssentia*)
- P2 Nivolumab / Pembrolizumab | PD-1 (*BMS / MRK*)

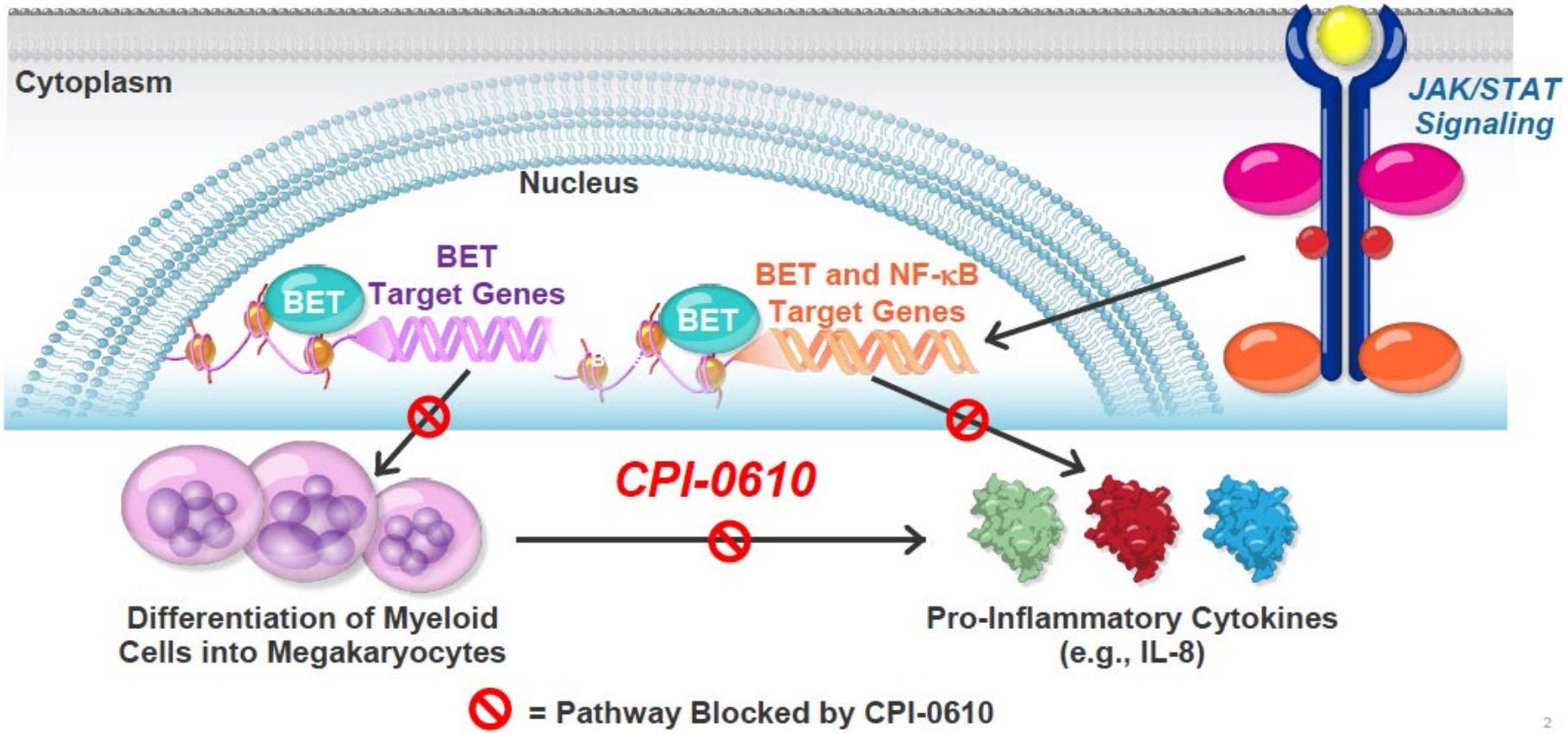
HDAC / Epigenetic

- P3 Azacytidine | HMA (*ESR/Celgene*)
- P3 Panobinostat | HDAC (*Novartis*)
- P2 Givinostat I HDAC (Italfama)
- P2 IMG-7289 | LSD1 (*Imago*)
- P1 CPI-0610 | BETi (*Constellation*)
- P1 PU-H71 | HSP90i (*Samus*)

PHASE OF DEVELOPMENT (IN MPN): P1 P2 P3

Mechanism of Potential Disease Modification in Myelofibrosis

Reduce Inflammation and Suppress Cells in the Bone Marrow That Drive MF (Megakaryocytes)



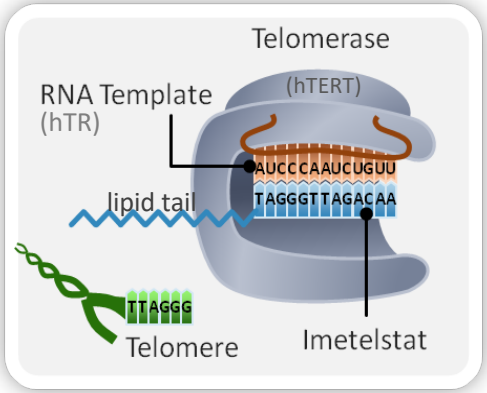
Mascarenhas et. al.
ASH 2019

Imetelstat: First-in-Class Telomerase Inhibitor

Imetelstat

- **Proprietary:** 13-mer thio-phosphoramidate oligonucleotide complementary to hTR, with covalently-bound lipid tail to increase cell permeability.
- **Potent, first in class competitive inhibitor of telomerase**: IC50 = 0.5-10 nM
- **Target:** selectively targets heme (MF) malignant stem and progenitor cell proliferation.[1, 2]

Imetelstat binds to RNA template preventing maintenance of telomeres



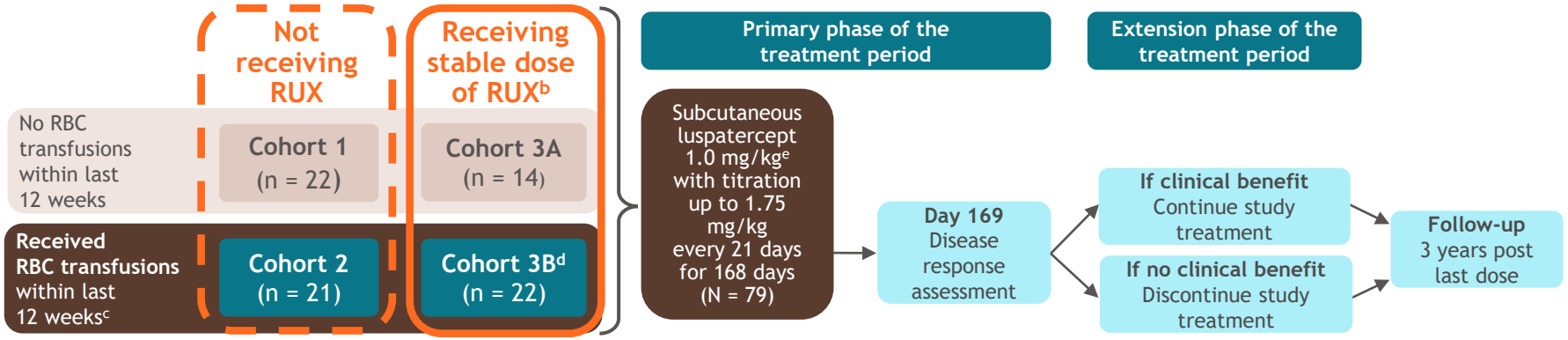
- Short telomere length (TL), high levels of telomerase activity (TA) and high expression of human telomerase reverse transcriptase (hTERT) correlated with higher risk, disease progression and shorter OS in patients with myeloid malignancies.[3-5]
- Nonclinical studies demonstrated that imetelstat reduces TA, hTERT expression level, and JAK2V617F^{+} hematopoietic progenitor cells in MF patient samples, indicative of mechanism based on-target activity.[1,2]
- **Cells with high levels of TA and hTERT and short TL, represent best target for treatment with telomerase inhibitor.**

[1]Wang, et al. *Blood Adv* 2018;2:2378-88.
[2]Mosoyan, et al. *Leukemia* 2017;31:2458-67.
[3]Briatore, et al. *Cancer Biol Ther* 2009;8:883-9.
[4]Kishtagari and Watts. *Ther Adv Hematol* 2017;8:317-26.
[5]Wang, et al. *Int J Lab Hematol* 2010;32:230-8.

ACE-536-MF-001 study design

- This study reports the results of the ongoing open-label, phase 2 ACE-536-MF-001 trial evaluating luspatercept in subjects with MF and anemia, focusing on response in subjects requiring RBC transfusions (NCT03194542)

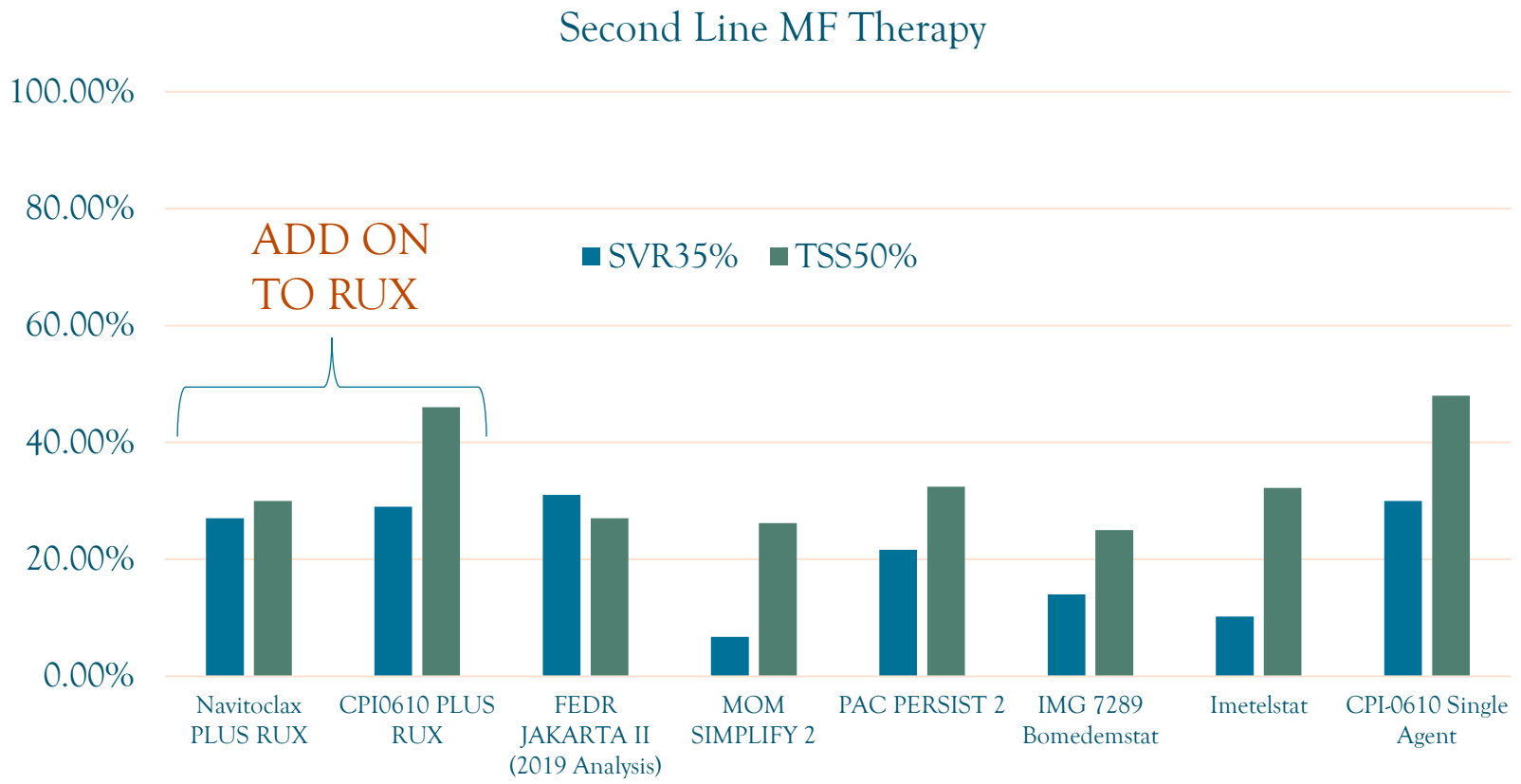
Figure 1. ACE-536-MF-001 study design[a]



- 79 subjects with MF and anemia had been enrolled by the data cutoff and were included in this updated analysis (March 29, 2020)
- The analyses presented here focus on response in subjects requiring RBC transfusions (Cohorts 2 and 3B); safety is reported for all 79 subjects on study

As of March 29, 2020, 16 (20%) subjects remain on treatment. [a]Enrolled subjects had primary or post-essential thrombocythemia/post-polycythemia vera myelofibrosis; [b]A stable daily dose of RUX for at least 16 weeks at enrollment; for the 3 subjects enrolled in the expansion cohort in Cohort 3B, subjects were receiving a stable RUX dose for 40 weeks; [c]6-12 RBC units/84 days prior to treatment; or 4-12 units/84 days for the 3 subjects enrolled in the expansion cohort in Cohort 3B; [d]Including 3 subjects enrolled in the expansion cohort; [e]The starting dose was 1.33 mg/kg in the expansion cohort subjects. MF, myelofibrosis; RBC, red blood cell; RUX, ruxolitinib.

Comparison for 2L Therapy



Pemmaraju et. al. ASH 2020
Verstovsek et. al. ASH 2020
Harrison et. al. ASH 2019
Verstovsek et. al.
Mascarenhas et. al.
Yacoub et. al. ASH 2020
Mascarenhas et. al. ASH 2020
Talpaz et. al ASH 2020

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MPNs 2021

- Burden of Having an MPN
- Essential Thrombocythemia
- Polycythemia Vera
- Myelofibrosis
- Complementary Approaches

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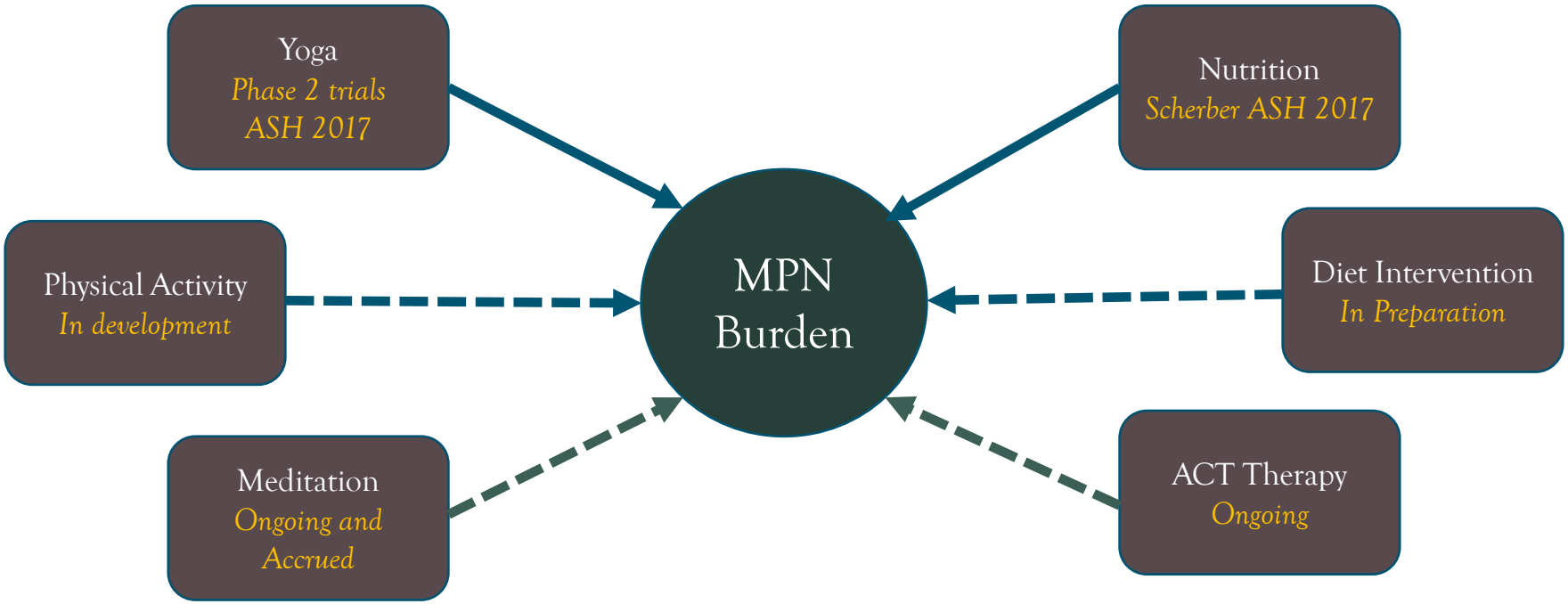
Non Pharmacological Approaches for MPN Burden Relief



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Conclusions
MPNs 2021

1) Adequately assess the burden on the MPN and develop appropriate therapy and goals

2) If therapy is not beneficial change to alternative therapy or clinical trial

3) JAK inhibitors and interferons do have a benefit for many subsets of MPN patients, yet opportunities exist

4) Multiple additional pathway targeted agents are undergoing parallel testing – primarily in 2nd line MF and 3rd line PV or ET

5) Non pharmacological therapies may augment treatment options for MPNs

MPN QoL STUDY GROUP

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