

# Updates in Diagnosis and Treatment of Myelodysplastic Syndromes

Aplastic Anemia and MDS Association of Canada

Patient and Family Support Group

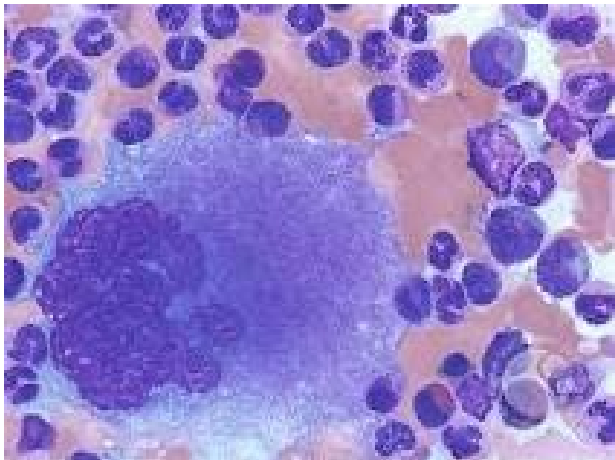
October 14, 2023

# Objectives

- Review diagnosis of MDS
- Update ways to evaluate prognosis of disease
- Overview the management of lower risk MDS
- Outline management of higher risk MDS

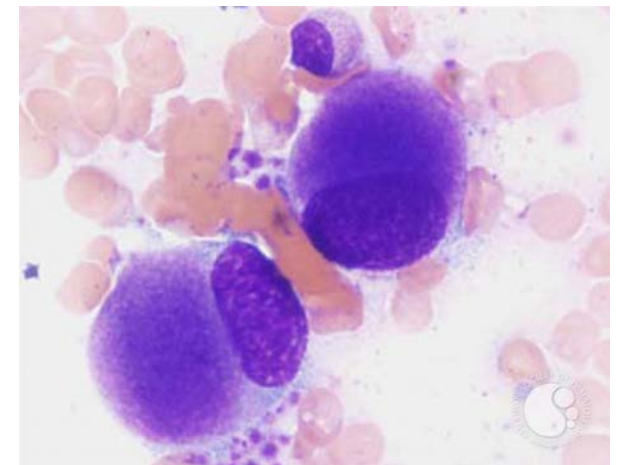
# What are myelodysplastic syndromes?

- A group of bone marrow disorders with the following characteristics:
  - Bone marrow failure
    - symptoms of low blood counts
  - Dysplastic (abnormal) blood cells under the microscope
  - Clonal mutation seen in chromosomes or gene mutations
  - Increased risk of developing acute myeloid leukemia



Normal megakaryocyte (platelet precursor)

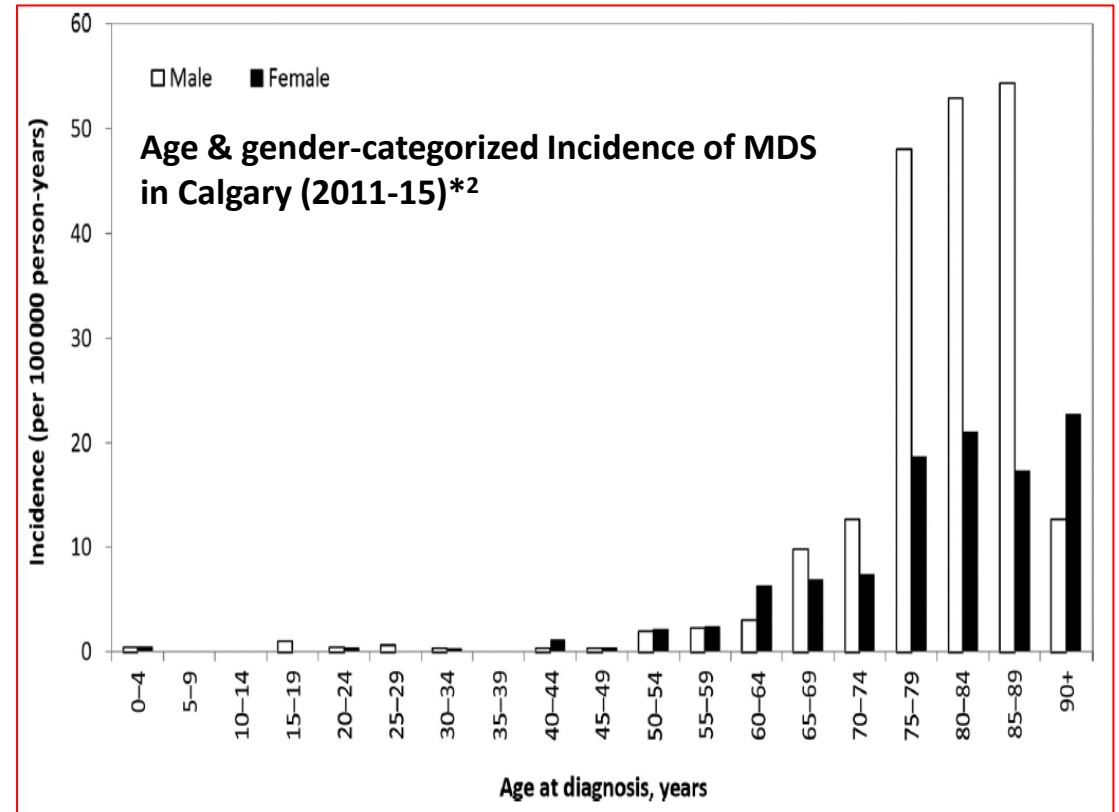
ASH image bank



Dysplastic megakaryocyte

# MDS in Canada

- MDS is likely under-diagnosed
- Prevalence: ~10,000 - 40,000 aged  $\geq 65$  yrs
- Incidence (new diagnosis): ~3,850 in 2016
- Retrospective cohort study from Calgary Metropolitan Area
- Male to female ratio: ~1.35
  - Median age of diagnosis: ~75 yrs



1. Blood Cancer in Canada Facts & Stats 2016, Accessed 29<sup>th</sup> Dec, 2020, Link: [https://www.llscanada.org/sites/default/files/National/CANADA/Pdf/InfoBooklets/Blood\\_Cancer\\_in\\_Canada\\_Facts\\_%26\\_Stats\\_2016.pdf](https://www.llscanada.org/sites/default/files/National/CANADA/Pdf/InfoBooklets/Blood_Cancer_in_Canada_Facts_%26_Stats_2016.pdf)
2. Incidence of Myelodysplastic Syndromes in a Major Canadian Metropolitan Area, Jonathan Slack et al, November 2018 | 03:03 | 378-383 | JALM 379
3. The Population Based Incidence of Myelodysplastic Syndrome (MDS): Utility of Multiple Data Sources and Follow-up, Rajat Kumar et al, Blood (2009) 114 (22): 245
4. Estimating the prevalence of myelodysplastic syndromes in patients with unexplained cytopenia: A retrospective study of 322 bone marrows, Rena Buckstein et al, Leukemia Research, Volume 33, Issue 10, October 2009, Pages 1313-1318

# What are known risk factors for developing MDS?

## Environmental exposures

Professional	Adjusted OR	95% CI	P
Petrol	2.5	(0.9–7.7)	0.8
Aromatic polycyclic hydrocarbons	1.8	(0.7–4.6)	0.93
Exhaust gases	1.0	(0.5–1.9)	0.27
Dyes	2.3	(0.5–13.9)	0.77
Plastic fumes and dusts	3.5	(0.7–34.5)	0.09
Wood dusts	1.0	(0.3–3.3)	0.71
Pesticides	3.2	(1.1–11.2)	0.21
Fertilizers	2.9	(1.2–8)	0.96
Cereal dust	2.6	(1.2–6)	0.58
Poultry	15	(2.3–631.5)	0.1
Cotton and flax dusts	3.4	(1.6–8.2)	0.013
Radiation	2.25	(1.1 - 4.7)	NR

## High risk professionals

Professional	Adjusted OR	95% CI	P
Agricultural workers	3.66	(1.9–7.0)	0.0001
Textile operators	3.66	(1.7–7.9)	0.001
Health professionals	10.0	(2.1–48.7)	0.004
Living next to an industrial plant	2.45	(1.5–4.1)	0.007
Commercial and technical sales representatives	4.45	(1.4–14.6)	0.013
Smoking	1.74	(1.1–2.7)	0.015
Machine operators	2.69	(1.2–6.0)	0.015
Oil use	1.10	(1.0–1.2)	0.029

# Symptoms of Anemia

- Fatigue
- Shortness of breath, especially with exertion
- Heart racing
- Lightheadedness
- Sometimes angina, especially with exertion
- Heart pounding in ears
- Pale



# Symptoms of low platelets

- Easy bruising
- Bleeding from mucous membranes:
  - Nosebleeds
  - Gum bleeding with brushing teeth
  - Hemorrhoids
  - Heavy periods



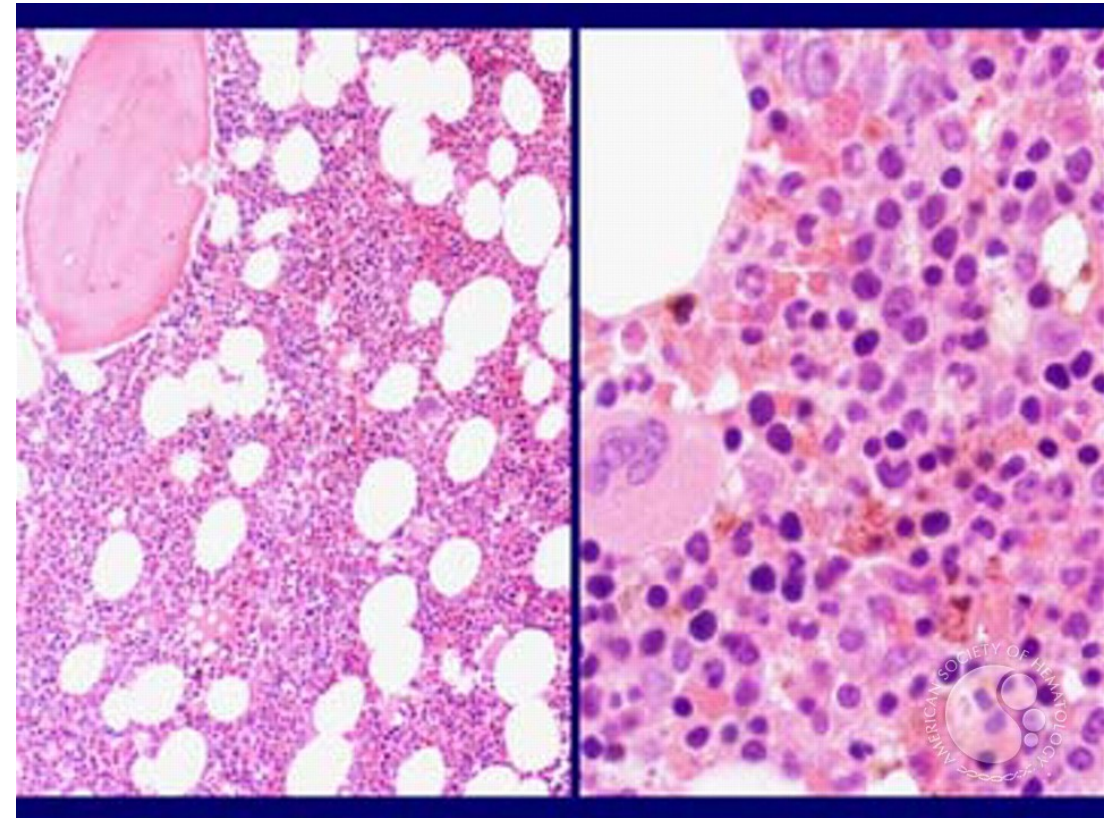
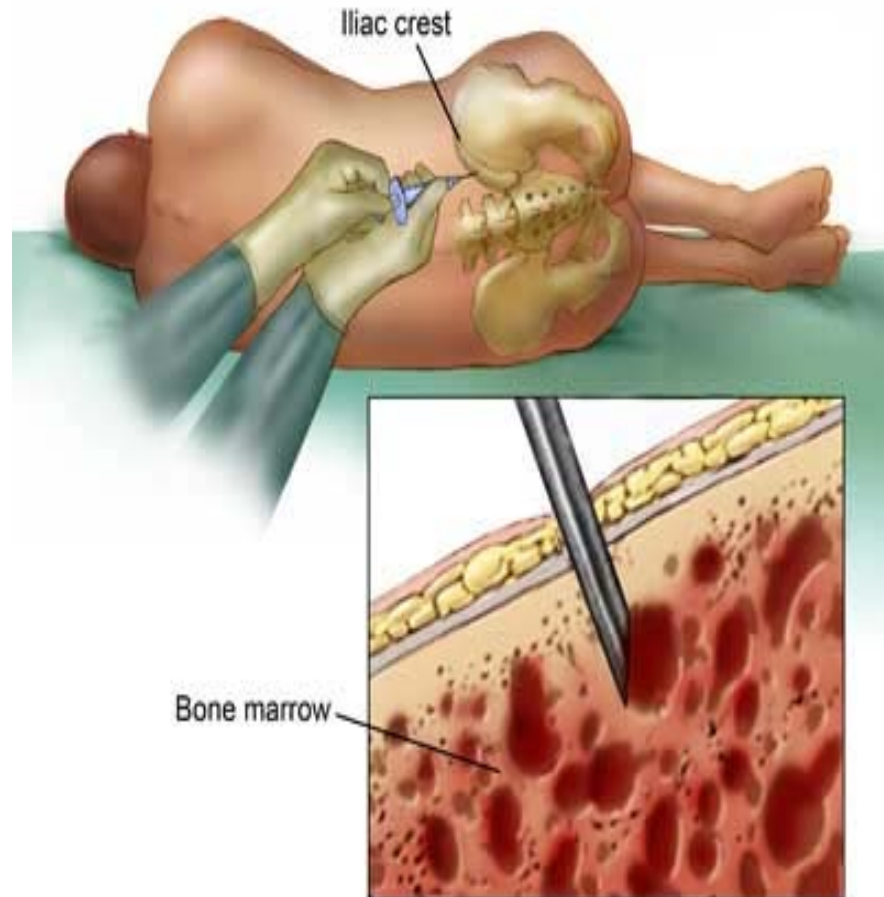
# Symptoms of low white blood cells

- Infections
  - More frequent
  - Higher risk of becoming more ill with infections





Gold standard test for diagnosis of MDS is a bone marrow biopsy



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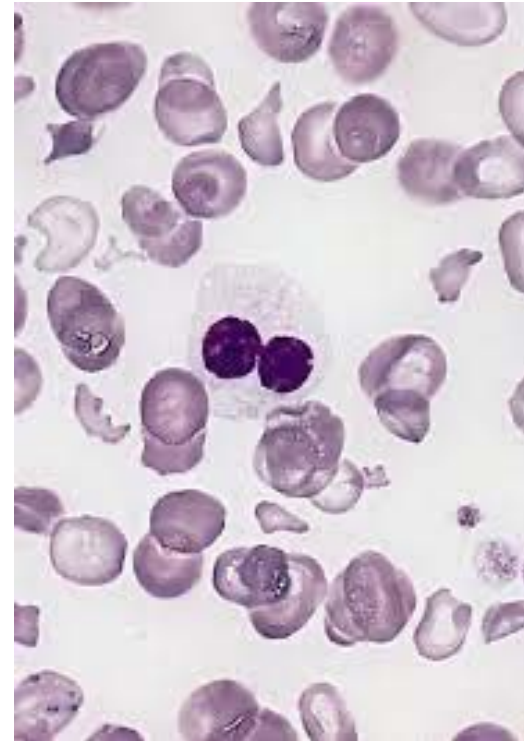
# What tests can be done on the marrow from a bone marrow biopsy?

## ➤ Microscopy

- What do the cells look like?
- Is the marrow empty or full?
- Are there increased numbers of immature blast cells?
  - Normal is less than 5%

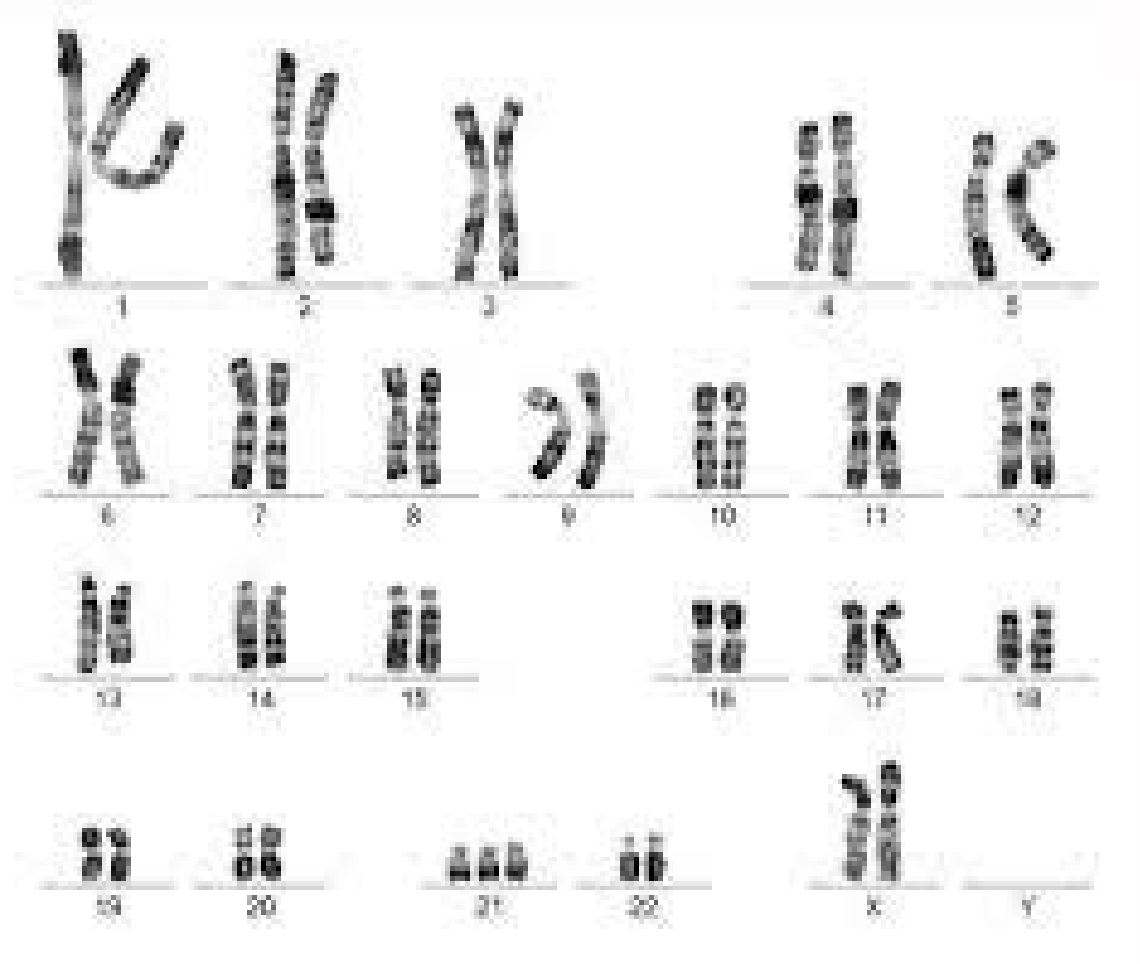
## • Flow cytometry

- Looks at patterns of surface markers on the cells



# What tests can be done on the marrow from a bone marrow biopsy?

- Cytogenetics



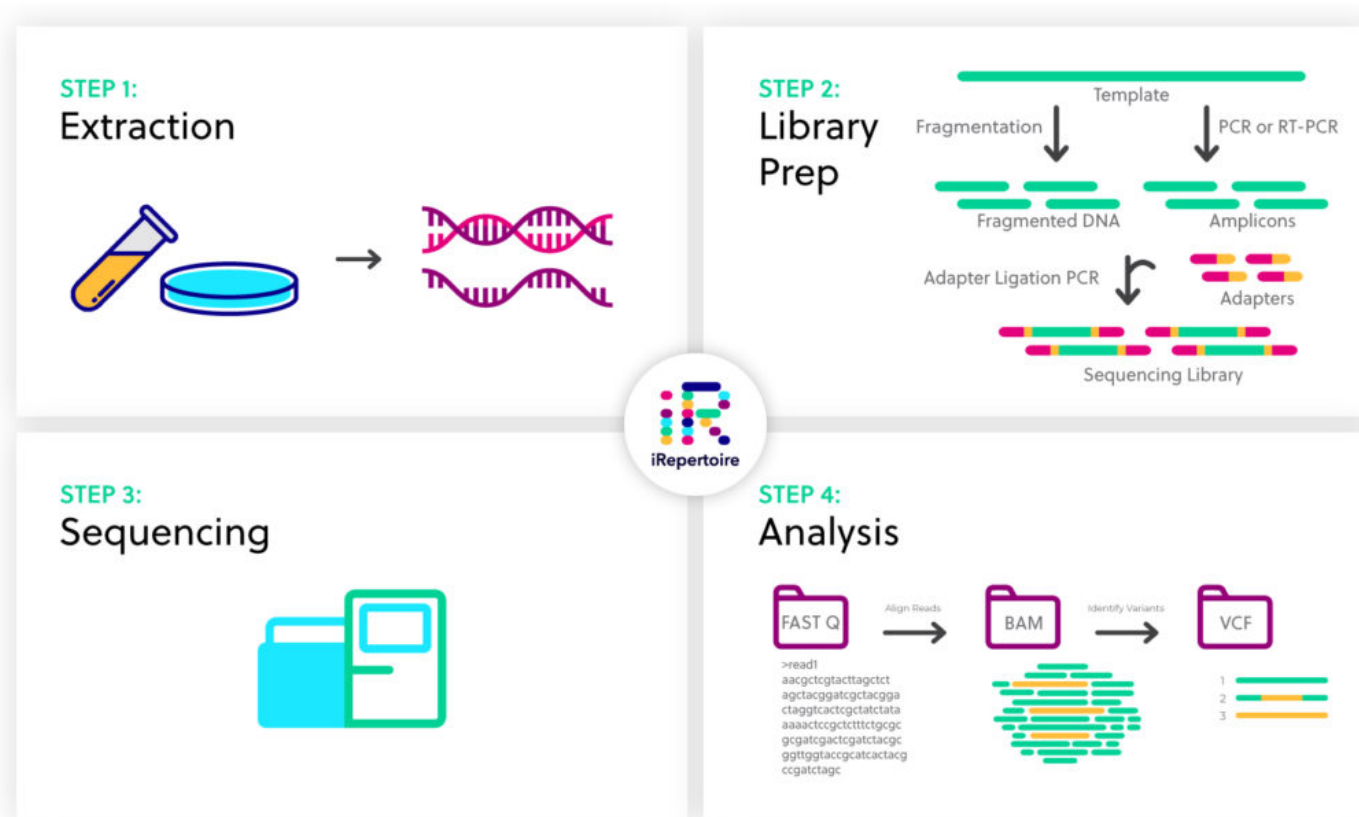
Some chromosome abnormalities are enough to make a diagnosis of MDS

Chromosomal abnormality	Frequency	
	MDS overall	Therapy-related MDS
<b>Unbalanced</b>		
Gain of chromosome 8 <sup>a</sup>	10%	
Loss of chromosome 7 or del(7q)	10%	50%
del(5q)	10%	40%
del(20q) <sup>a</sup>	5–8%	
Loss of Y chromosome <sup>a</sup>	5%	
Isochromosome 17q or t(17p)	3–5%	25–30%
Loss of chromosome 13 or del(13q)	3%	
del(11q)	3%	
del(12p) or t(12p)	3%	
del(9q)	1–2%	
idic(X)(q13)	1–2%	
<b>Balanced</b>		
t(11;16)(q23.3;p13.3)		3%
t(3;21)(q26.2;q22.1)		2%
t(1;3)(p36.3;q21.2)	1%	
t(2;11)(p21;q23.3)	1%	
inv(3)(q21.3q26.2) / t(3;3)(q21.3;q26.2)	1%	
t(6;9)(p23;q34.1)	1%	
<sup>a</sup> As a sole cytogenetic abnormality in the absence of morphological criteria, gain of chromosome 8, del(20q) and loss of Y chromosome are not considered definitive evidence of MDS; in the setting of persistent cytopenia of undetermined origin, the other abnormalities shown in this table are considered presumptive evidence of MDS, even in the absence of definitive morphological features.		

In the presence of unexplained cytopenias, these chromosome abnormalities are diagnostic of MDS. Del (20q), +Y and +8 are not diagnostic of MDS.

# What tests can be done on the marrow from a bone marrow biopsy?

- Mutations (NGS = next generation sequencing)



# MDS is can have many different gene mutations

- DNA damage and evolution of clonal cytogenetic abnormalities
  - Frequent abnormalities in DNA methylation and epigenetics

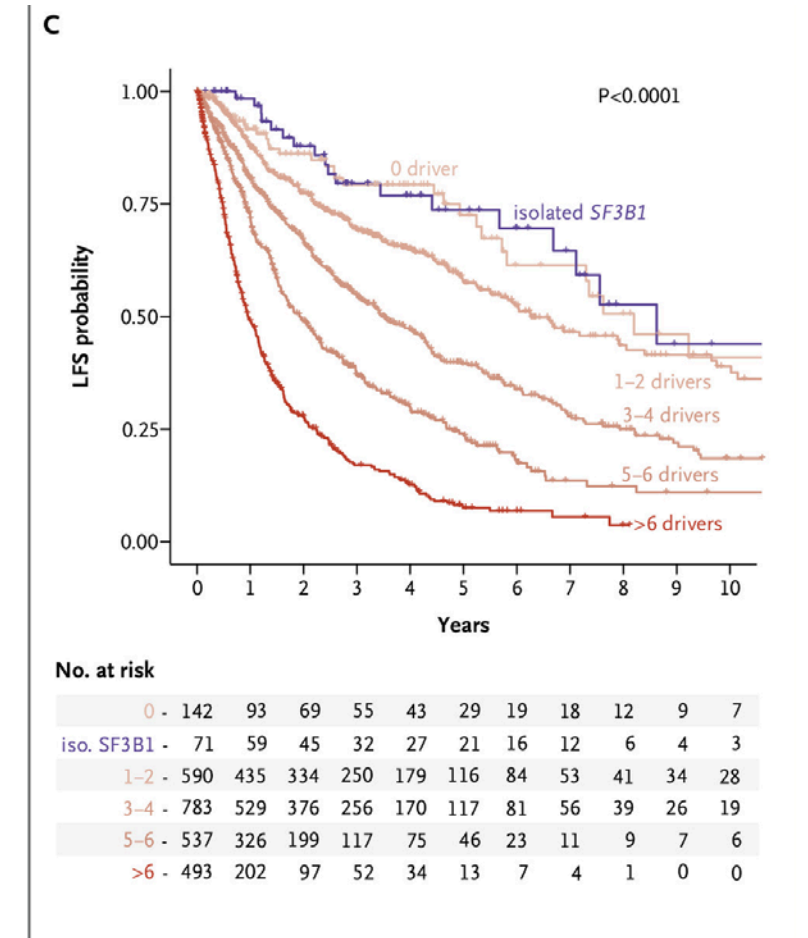
RARa

Signalling	Epigenetic	mRNA Splicing	Transcription	Cohesin genes	DNA damage
Jak2 CBL NRAS PTPN11 BRAF GNAS	TET2 DNMT3A IDH1/2 EZH2 ASXL1	SF3B1 SRSF2 U2AF1/2 ZRSR2	RUNX1 ETV6 BCOR CEBPA GATA2	STAG2 SMC1A SMC3 RAD21	TP53



# Driver mutations in MDS

89% of patients have a somatic mutation  
Typical MDS patient has 2-3 mutations



# MDS World Health Organization Classification 2022

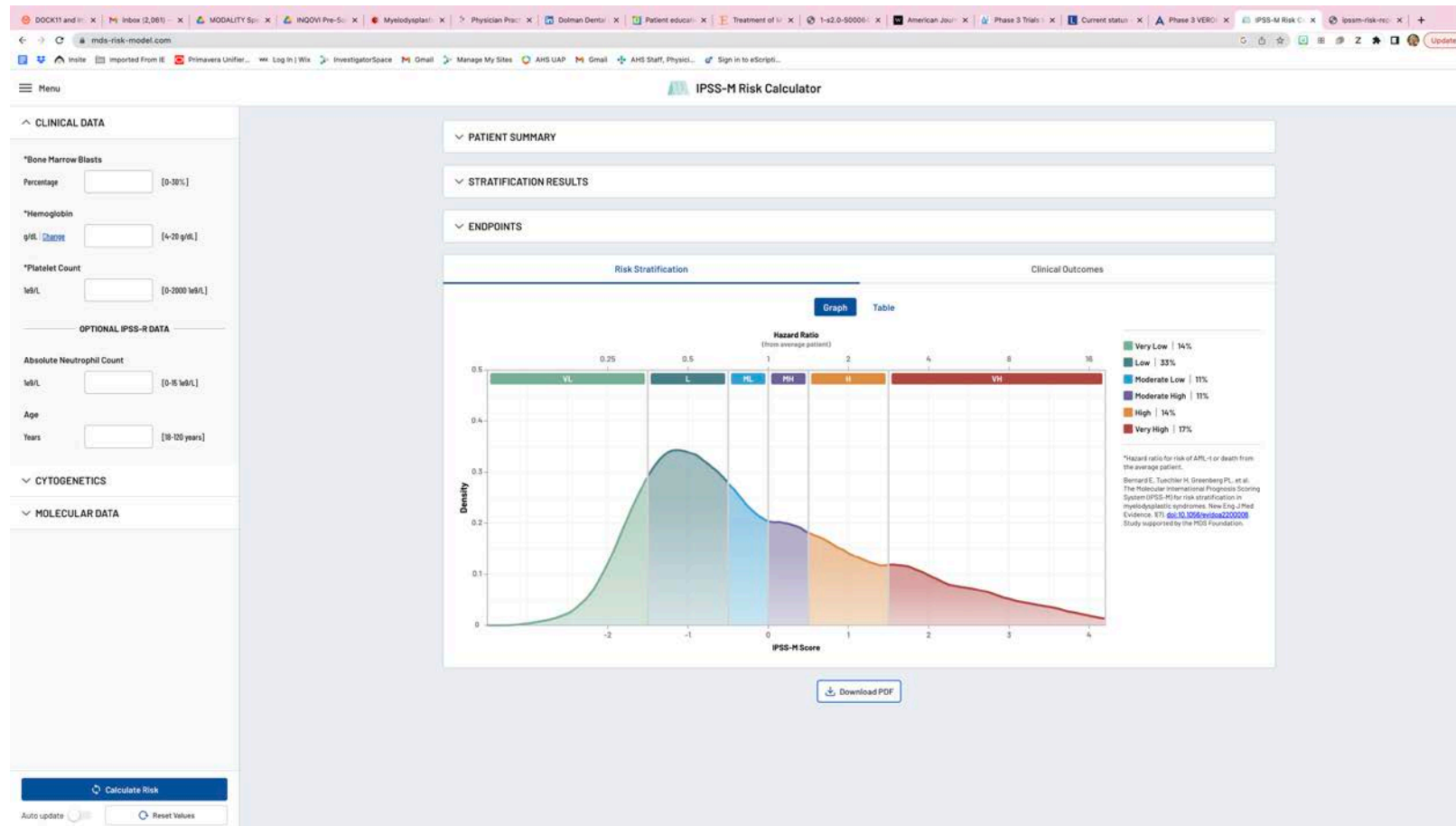
- **MDS with defining genetic abnormalities**
  - MDS with low blasts and isolated 5q deletion (**MDS-5q**)
  - MDS with low blasts and *SF3B1* mutation (**MDS-*SF3B1***)<sup>‡</sup>
  - MDS with biallelic *TP53* inactivation (**MDS-bi*TP53***)
- **MDS, morphologically defined**
  - MDS with low blasts (**MDS-LB**)
  - MDS, hypoplastic<sup>§</sup> (**MDS-h**)
  - MDS with increased blasts (**MDS-IB**)
    - MDS with increased blasts 1 (MDS-IB1) (5-9% BM or 2-4% PB)
    - MDS with increased blasts 2 (MDS-IB2) (10-19% BM or 5-19% PB or Auer rods)
    - MDS with fibrosis (MDS-f) (5-19% BM; 2-19% PB)



# R-IPSS score prognosticates based on blood counts, marrow blast count and cytogenetic risk

Risk Group	Points	% of Patients	Median Survival, Yr	Time Until 25% of Patients Develop AML, Yr
Very low	≤1.5	19%	8.8	Not reached
Low	>1.5-3	38%	5.3	10.8
Intermediate	>3-4.5	20%	3.0	3.2
High	>4.5-6	13%	1.6	1.4
Very High	>6	10%	0.8	0.73

# IPSS-M Prognostic scoring system

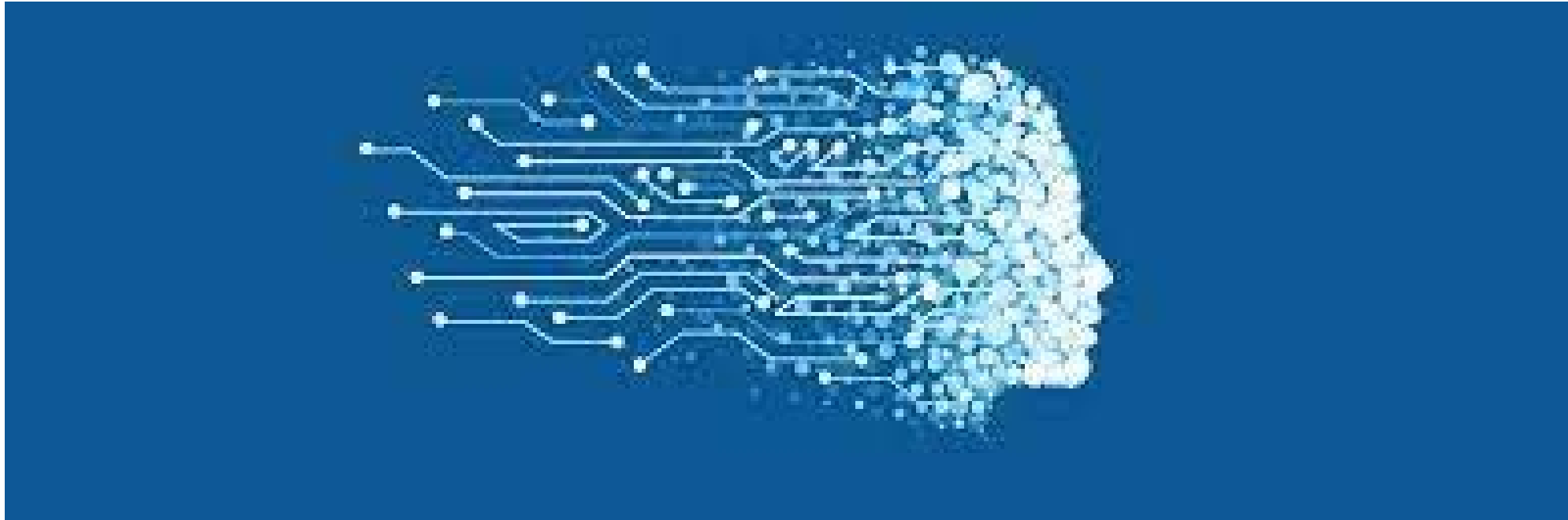


$$\text{score} = \frac{1}{\log 2} \sum_{\text{variables } j} w_j (x_j - x_j^{\text{mean}}) \quad 223$$

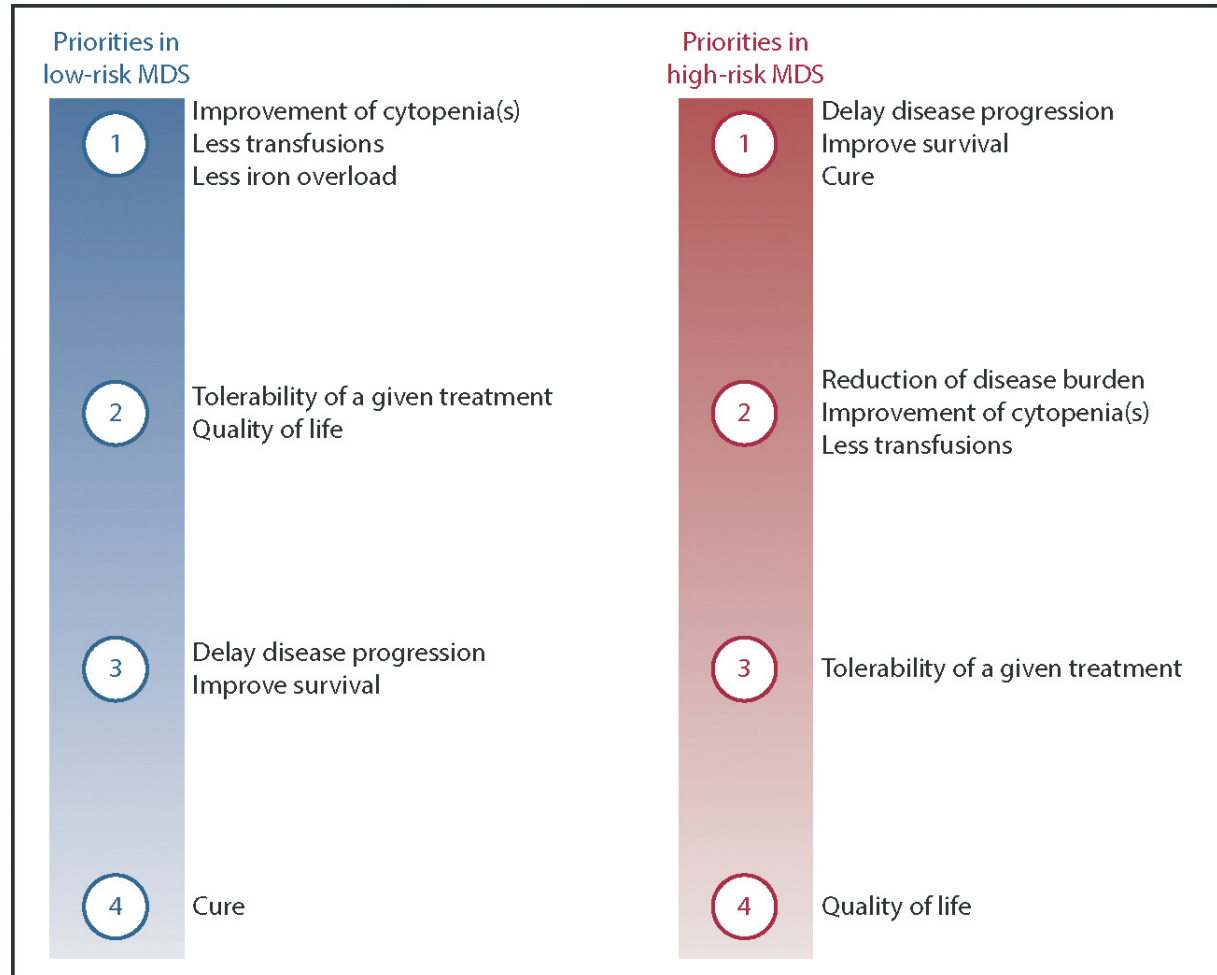
prognostic scoring  
group compared to the IPSS-R

Valid for therapy-related or  
secondary MDS that  
arises out of an underlying  
bone marrow disorder

# Machine Learning may help incorporate MDS molecular risk factors into prognostic scores



# Goals in care of patients with MDS



# Treatment Options

- Supportive Care
- Transfusions
- Antibiotics
- Iron chelation
- Growth Factors
- Hypomethylating agents
- Lenalidomide
- Immunosuppression
- Chemotherapy
- Bone marrow transplantation



# Supportive care

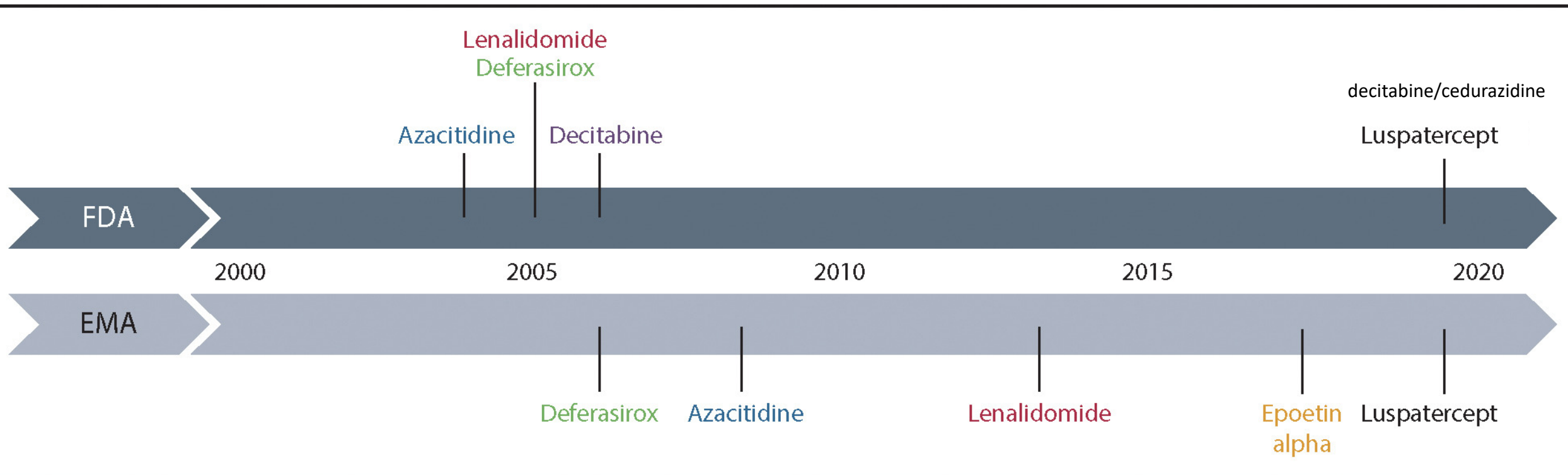
- Goal is to improve quality of life and also survival
  - Antibiotics
  - Transfusions of red cells and platelets
  - Home care support, mobile labs
  - Medications to prevent bleeding
  - Avoid iron supplements unless iron deficient





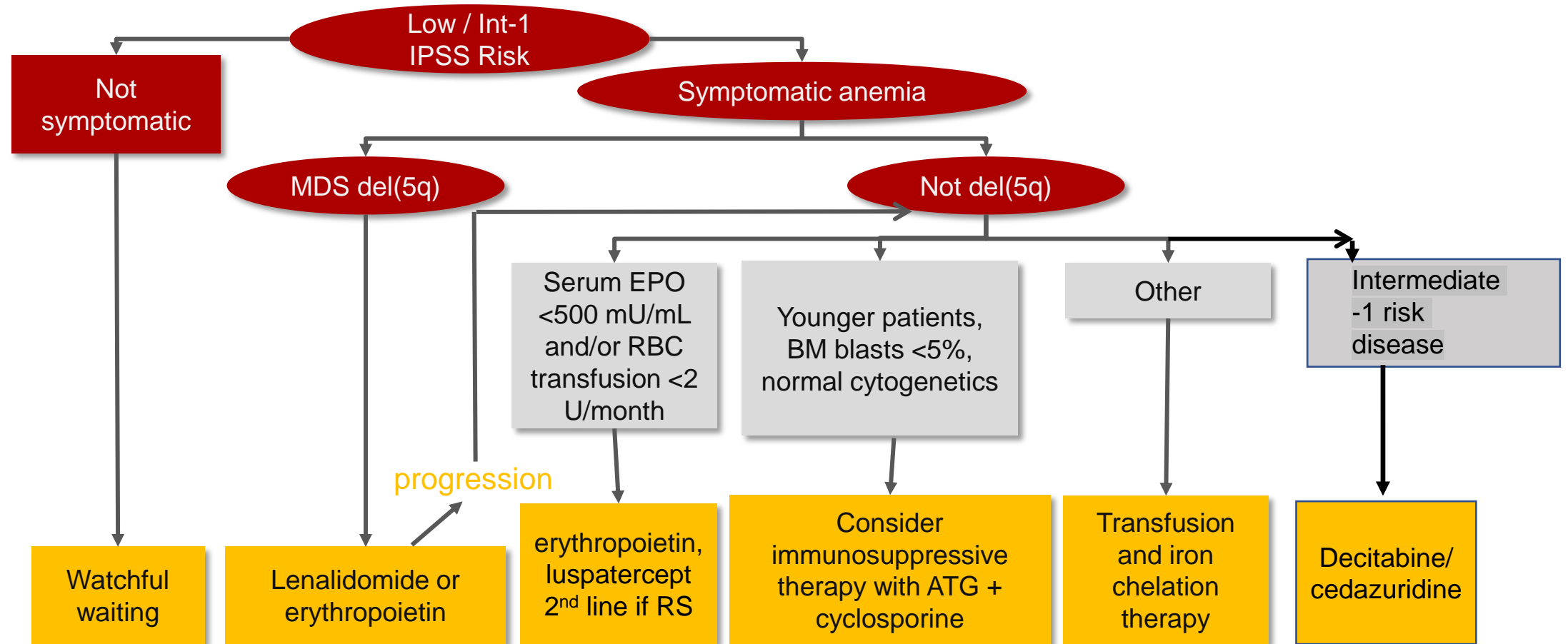
**"Satisfied NOW?!"**

# History of MDS treatments





# Recommendations for patients with low / intermediate-1 IPSS risk



# Growth factors for red cells

- Erythropoietin or darbopoietin
- Luspatercept

# What are erythropoietin and darbopoietin?

- Synthetic version of a hormone the kidney makes to stimulate the bone marrow to make more red cells
- First step for managing anemia in most patients with lower risk MDS.
- No difference between erythropoietin and darbepoietin in effectiveness
  - Weekly injection vs three week injection
- Start with a 8-12 weeks trial, can increase the dose
- Among responders average duration of response 12-18 months.



# Predictors of response to ESAs

Prognostic Factor	Score
Serum EPO > 200 mU/mL	1
Serum ferritin > 350 ng/mL	1
IPSS-R	
Very low	0
Low	1
Intermediate	2
High	3



Score	HI-E Rate (%)
0	85
1	80
2	64
3	40
4	20

# ITACA SCORE BETTER PREDICTS RESPONSE TO ESAs

A predictive scoring system of ESA response, ITACA, was developed using 3 large international ESA datasets, including a **Canadian database**<sup>1</sup>

The scoring system takes the following factors into consideration<sup>1</sup>:

- RBC transfusion independence (<1 unit/8 weeks x 16 weeks)
- Serum EPO concentration (<100 U/L)
- IPSS low risk

Each covariate was assigned a value of 1 resulting in a scoring system of 0 – 3<sup>1</sup>

The ITACA score has improved discriminatory capacity for predicting ESA response compared to the Nordic, **MDS-CAN** and IPSS-R based scoring systems<sup>1</sup>

**Response rate according to ITACA score in the derivation and validation cohorts<sup>1</sup>**

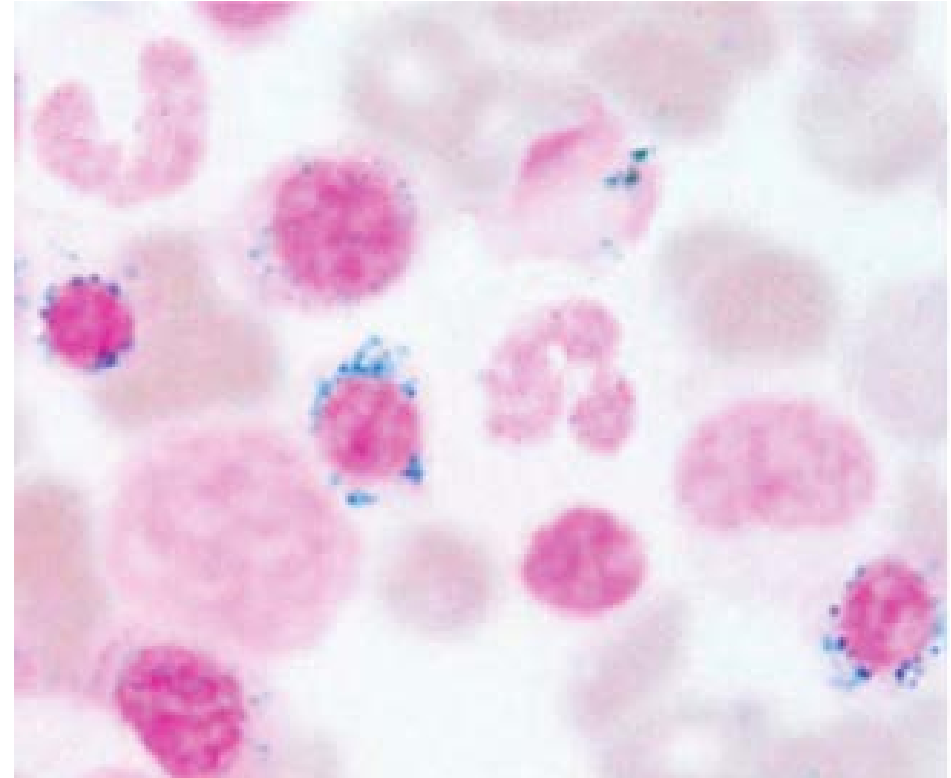
ITACA Score	Overall Response Rate (%)	
	Derivation Set (N=345)	Validation Set (N=336)
0	23%	22%
1	43%	42%
2	67%	64%
3	85%	78%

EPO, erythropoietin; ESA, erythropoiesis stimulating agent; IPSS, International Prognostic Scoring System; IPSS-R, International Prognostic Scoring System - Revised; RBC, red blood cell

<sup>1</sup>Buckstein R, et al. Am J Hematol. 2017;92(10):1037-1046.

# Role of SF3B1 in MDS diagnosis

- SF3B1 is involved in splicing pre-messenger RNAs (pre-mRNAs) that regulate key pathways in hematopoietic stem and progenitor cells associated with MDS
- Mutations in *SF3B1* are the most commonly associated with MDS, and are more prevalent in low-risk patients
  - **>70%** of MDS with ringed sideroblast patients have a mutation in *SF3B1*
- *SF3B1* mutations have been shown to be an independent predictor of favourable outcomes for MDS patients



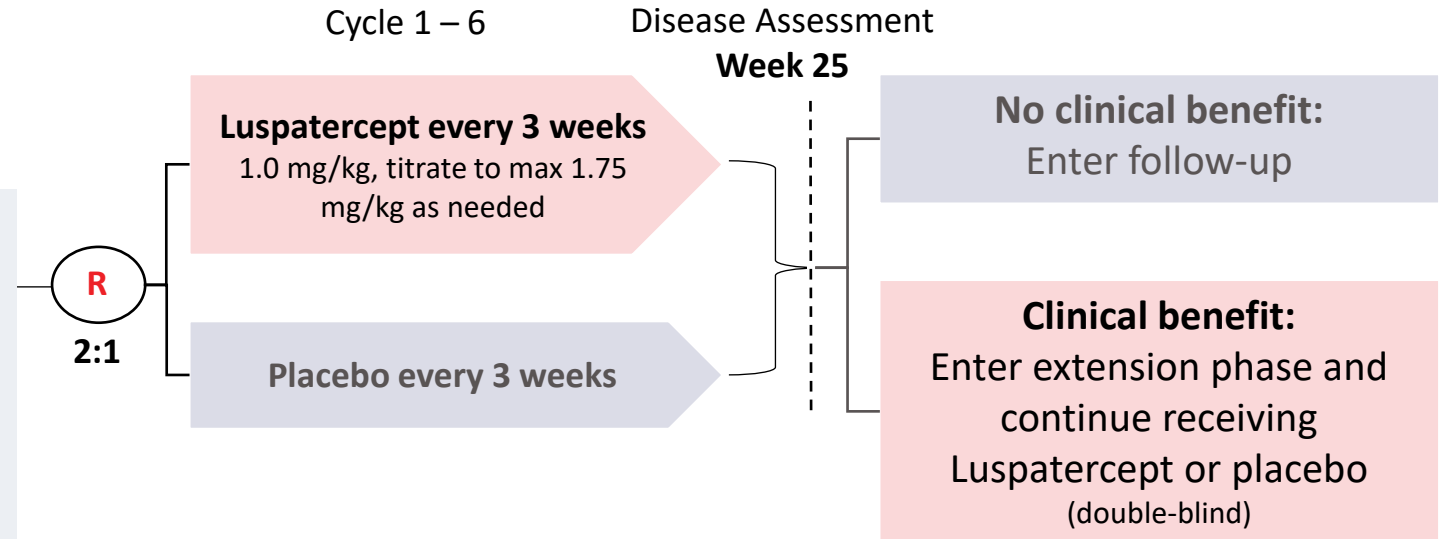
Ring sideroblasts in a bone marrow smear visualized through Perl's reaction. Adapted from Cazzola M, Invernizzi R. Haematologica. 2011;96(6):789-792.

# MEDALIST STUDY DESIGN

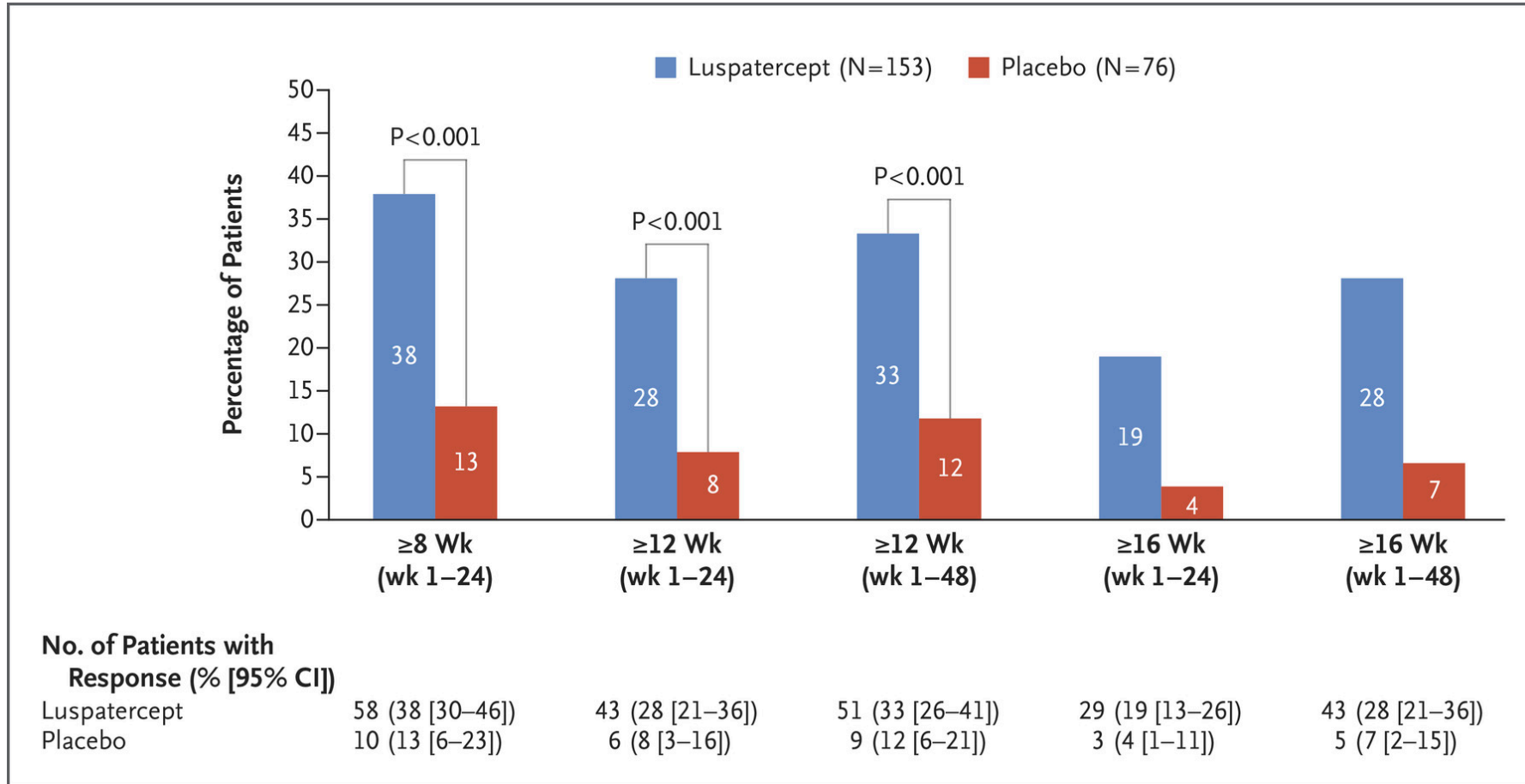
**MEDALIST was a double-blind, randomized, placebo-controlled, phase 3 study**

## Key Eligibility Criteria:

- Age  $\geq 18$  years
- MDS-RS (WHO):  $\geq 15\%$  RS or  $\geq 5\%$  with *SF3B1* mutation
- $< 5\%$  blasts in bone marrow
- No del(5q) MDS
- IPSS-R very low-, low-, or intermediate-risk
- Prior ESA response:
  - Refractory, intolerant
  - ESA naïve: EPO  $> 200$  U/L
- Average RBC transfusion burden  $\geq 2$  units/8 weeks
- No prior treatment with disease-modifying agents



# Transfusion independence rates luspatercept vs placebo RCT - MEDALIST



- RBC-TI\* for ≥8 weeks **38%** of patients on luspatercept vs **13%** on placebo ( $p<0.001$ )
- 62% (36/58) had ≥ 1 episode of RBC-TI during the treatment period
- Median duration of longest period of transfusion independence 30 weeks



# Luspatercept in MDS

- Luspatercept is approved for transfusion-dependent MDS with very low to intermediate risk (IPSS-R) MDS with ringed sideroblasts who have failed or are unsuitable for erythroid stimulating agents
- Application for Blue Cross Special Authorization required
  - 1 dose luspatercept per prescription from their pharmacy
- Reassessment every 6 months:
  - Red cell transfusion independent over minimum of 16 consecutive weeks over the previous 24 weeks

# The COMMANDS study

Phase 3, open-label, randomized trial comparing the efficacy and safety of luspatercept versus epoetin alfa for the treatment of anemia due to IPSS-R lower risk-MDS in ESA-naïve patients who require transfusions

## Key eligibility criteria

- ≥ 18 years of age
- IPSS-R Very low-, Low-, or Intermediate-risk MDS (with or without RS) by WHO 2016, with < 5% blasts in bone marrow<sup>a</sup>
- Required RBC transfusions (2–6 pRBC units/8 weeks for a minimum of 8 weeks immediately prior to randomization)
- Endogenous sEPO < 500 U/L
- ESA-naïve

Randomized  
1:1

**Luspatercept (N = 178)**  
1.0 mg/kg s.c. Q3W  
titration up to 1.75 mg/kg

**Epoetin alfa (N = 178)<sup>b</sup>**  
450 IU/kg s.c. QW  
titration up to 1050 IU/kg

**Response assessment at  
day 169 and every  
24 weeks thereafter**

**End treatment**  
Due to lack of clinical benefit<sup>c</sup>  
or disease progression

## Post-treatment safety follow-up

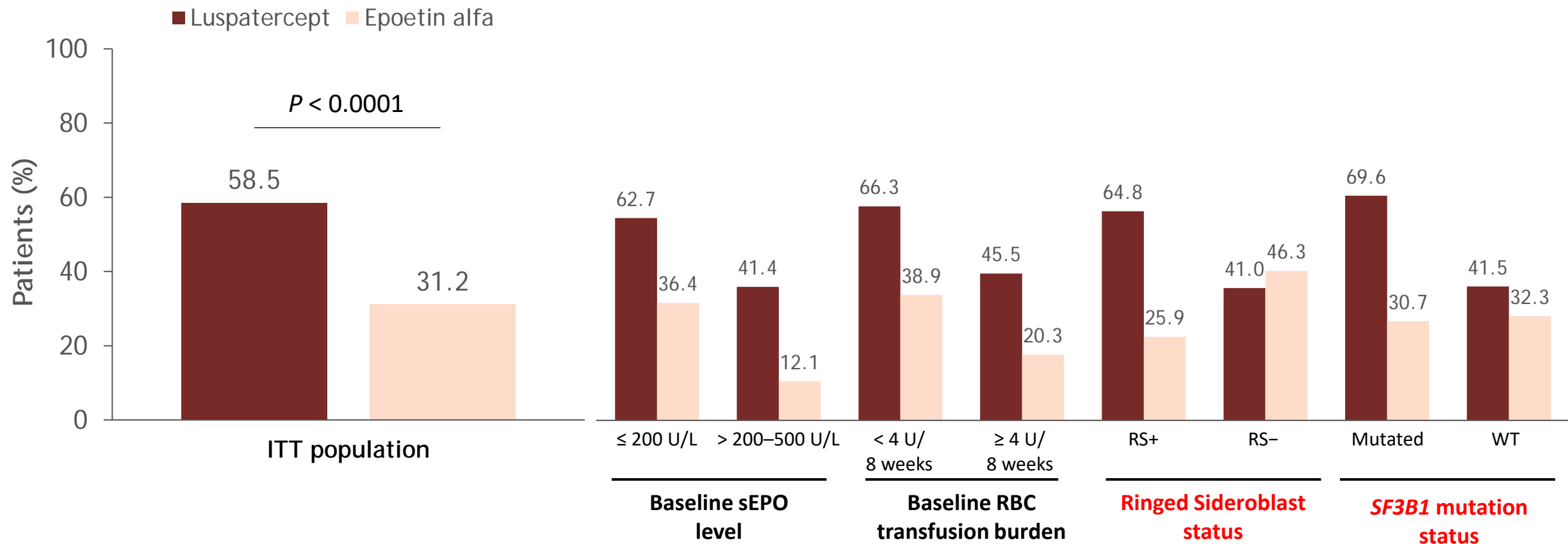
- Monitoring for other malignancies, progression to high risk-MDS or AML, subsequent therapies, survival

**Main endpoints: Transfusion independence and hematologic improvement**

<sup>a</sup>MDS with del(5q) were excluded; <sup>b</sup>2 patients randomized to the epoetin alfa arm withdrew consent prior to receiving their first dose; <sup>c</sup>Clinical benefit defined as transfusion reduction of ≥ 2 pRBC units/8 weeks versus baseline. AML, acute myeloid leukemia; HR, high risk; IPSS-R, Revised International Prognostic Scoring System; IWG, International Working Group; pRBC, packed RBC; QW, once weekly; Q3W, every 3 weeks; RBC, red blood cell; s.c., subcutaneously; sEPO, serum erythropoietin; WHO, World Health Organization.

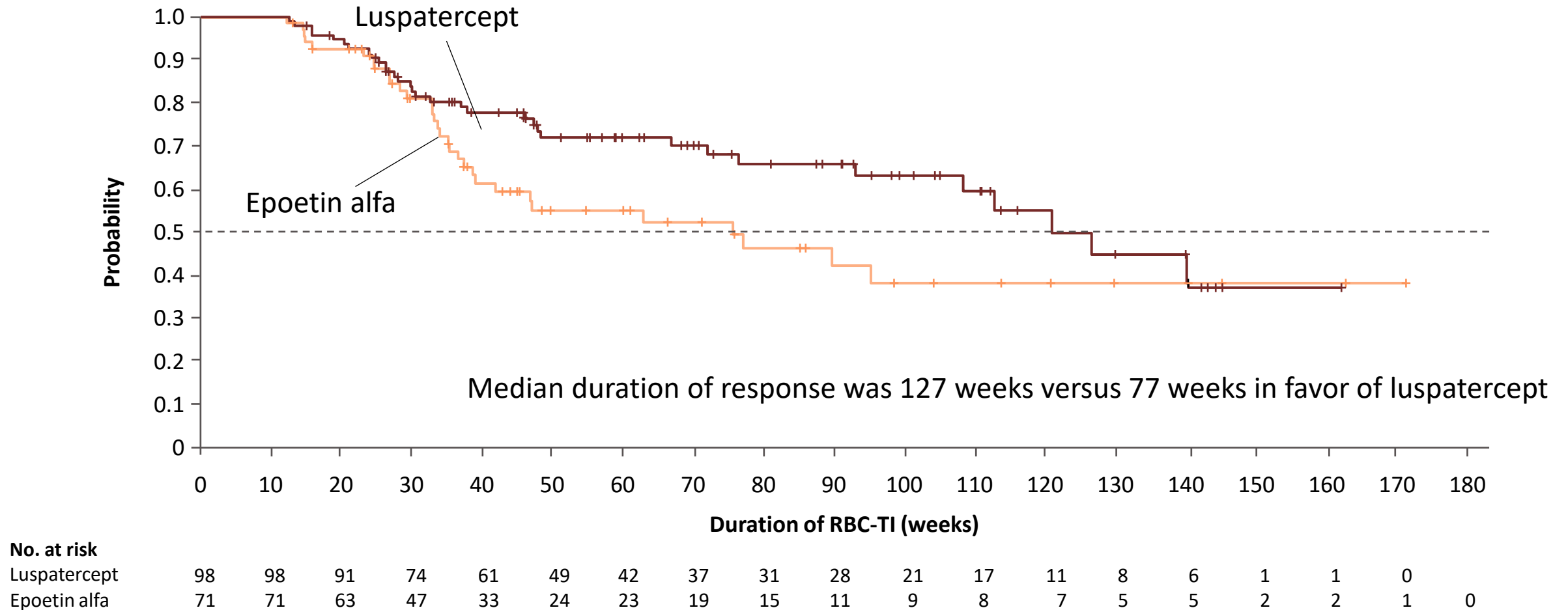
# COMMANDS: achievement of primary endpoint in different patient subgroups

- Primary endpoint: red cell transfusion independence  $\geq 12$  weeks with concurrent mean hemoglobin increase  $\geq 1.5$  g/dL (weeks 1–24)



RS status, baseline sEPO level, and baseline RBC transfusion burden were prespecified factors for randomization. *SF3B1* mutation status was a post hoc subgroup analysis. WT, wild type.

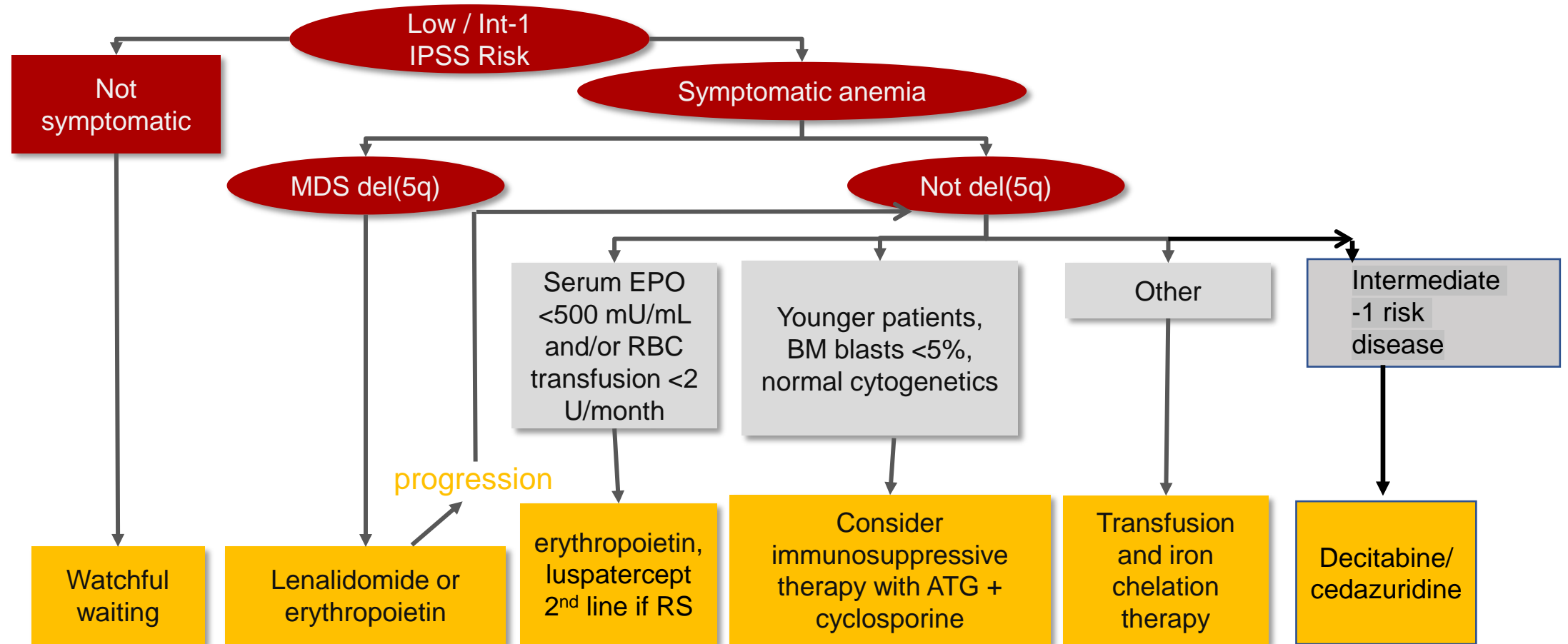
# Transfusion independence $\geq 12$ weeks in luspatercept and epoetin alfa patients



During week 1–EOT. CI, confidence interval; HR, hazard ratio; NE, not estimable.

Garcia-Manero G, et al. ASCO 2023 [Abstract #7003]

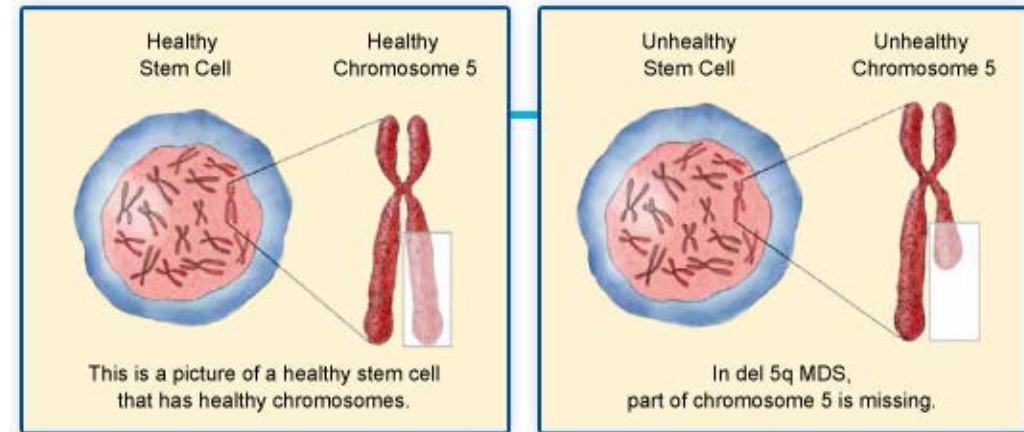
# Recommendations for patients with low / intermediate-1 IPSS risk



# Lenalidomide in patients with del(5q) MDS

- 10-15% of MDS
  - Anemia
  - Mild low white blood cells
  - Atypical megakaryocytes, normal to elevated platelets
  - Transfusion dependence
  - Normal blast count

Extended survival with low  
frequency of  
AML transformation

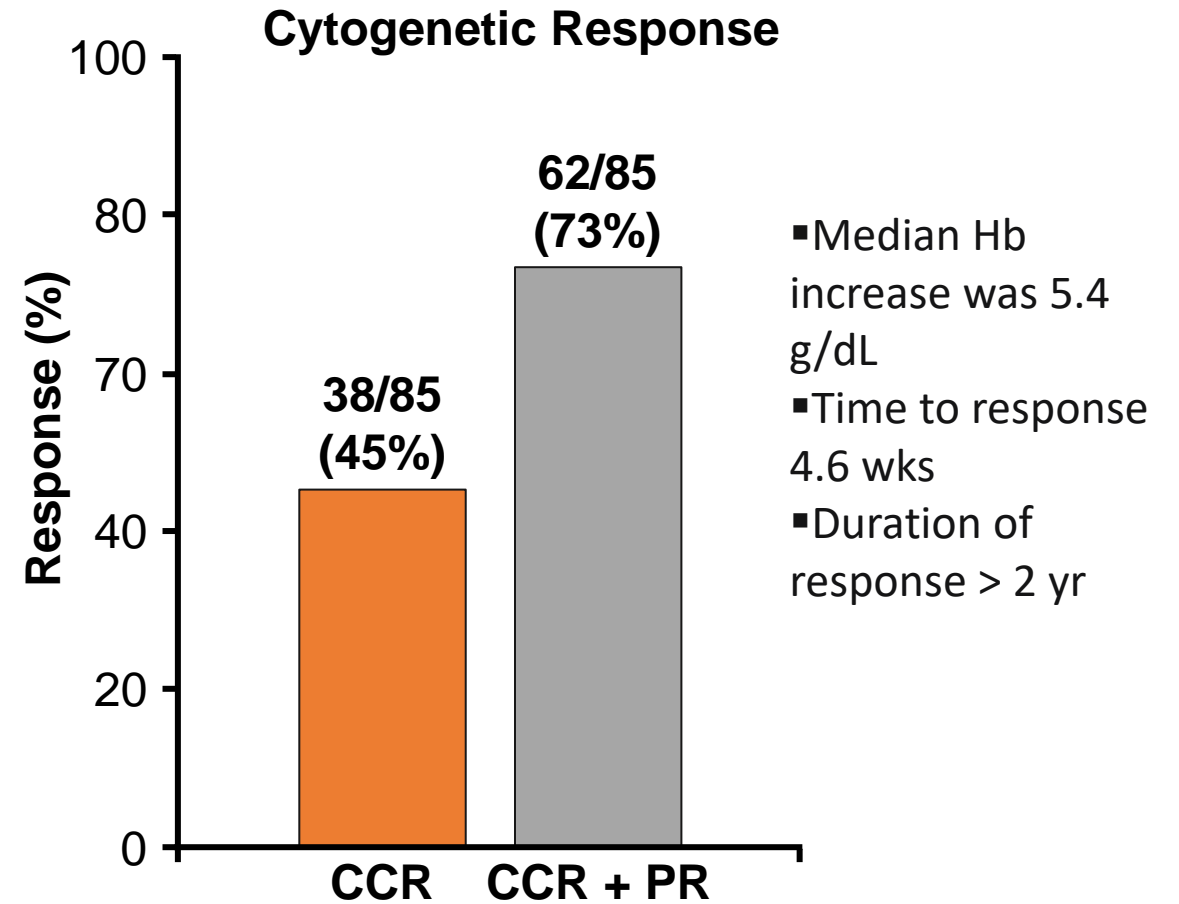
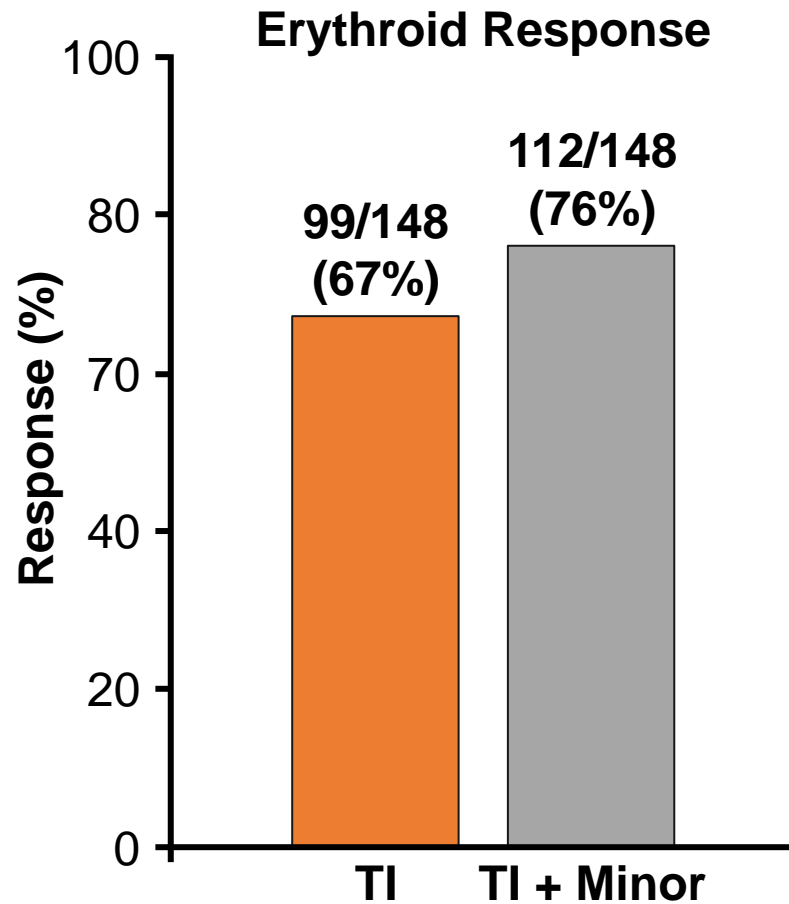


# Lenalidomide: RBC Transfusion Independence in Del(5q) MDS

<b>Hemoglobin Response Rate (N=148)</b>	<b>n (%)</b>	<b>95% CI</b>
<b>Transfusion independence*</b>	<b>99 (67)</b>	<b>59–74</b>
<b>≥50% decrease in no. transfusions</b>	<b>13 (9)</b>	<b>5–15</b>
<b>Total transfusion response</b>	<b>112 (76)</b>	<b>68–82</b>
<b>Transfusion Independence Response Characteristics</b>	<b>Median</b>	<b>Range</b>
<b>Time to response (wk)</b>	<b>46</b>	<b>1–49</b>
<b>Hgb increase<sup>†</sup> (g/dL)</b>	<b>54</b>	<b>11–114</b>

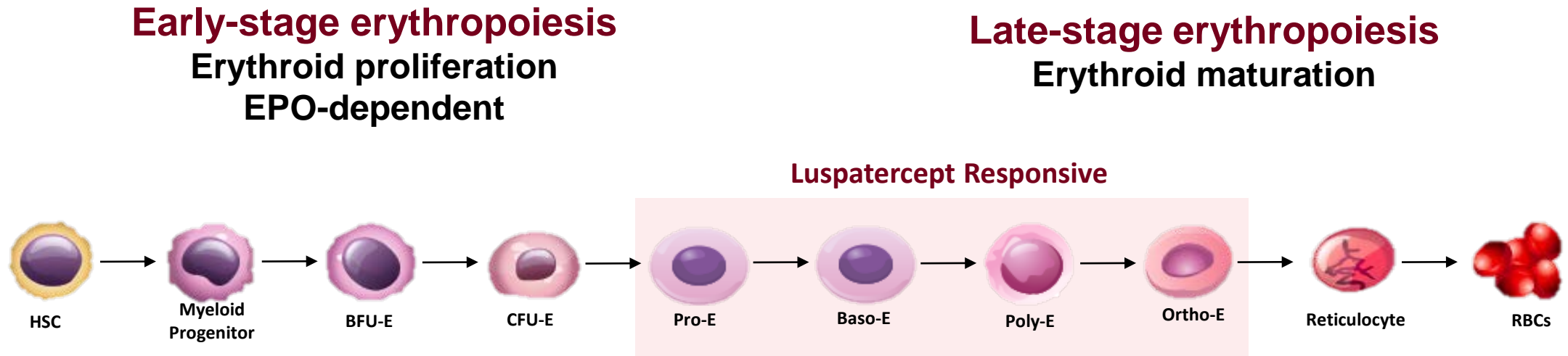
Can cause lower platelets and white cells, diarrhea, rash

# Response to Lenalidomide Therapy





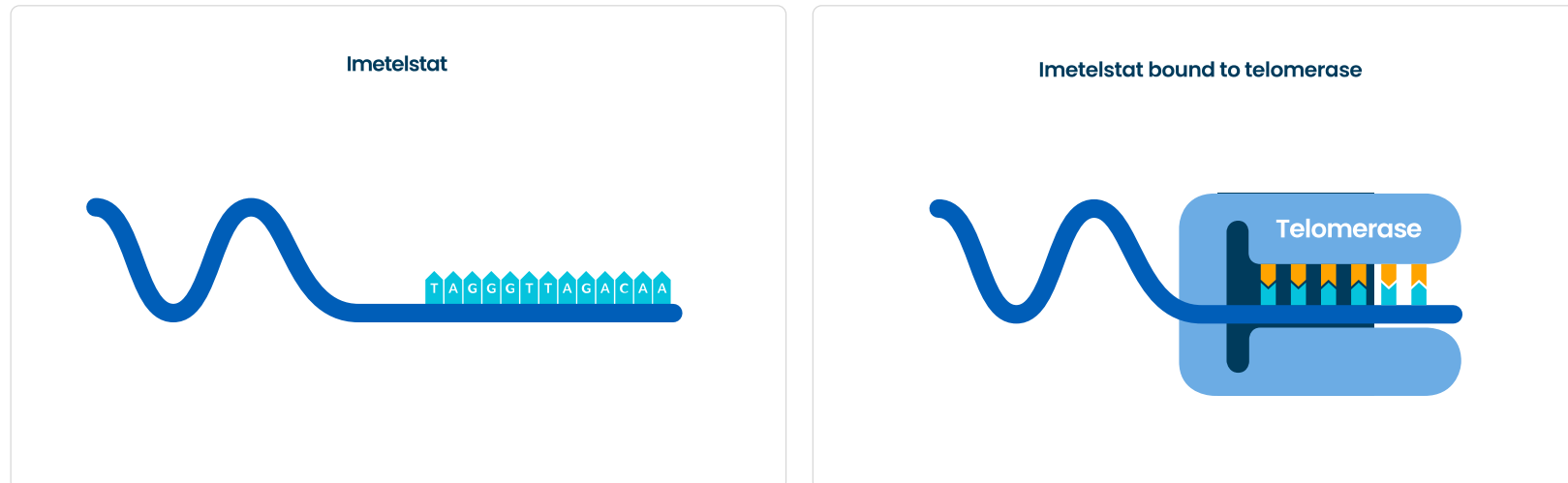
# Luspatercept targets late stage red cell production



Luspatercept is an erythroid maturation agent that binds to select TGF- $\beta$  superfamily ligands to inhibit aberrant Smad2/3 signalling and enhance late-stage erythropoiesis

# Coming?

- Imetelstat is a first in class telomerase inhibitor
- Telomerase are at the end of the DNA strands and get shorter with age
  - Shorter in MDS patients
- Oligonucleotides (short single strands of synthetic DNA or RNA) inhibit the activity of telomerase, an enzyme that helps maintain telomeres and enables the continued proliferation of malignant cells



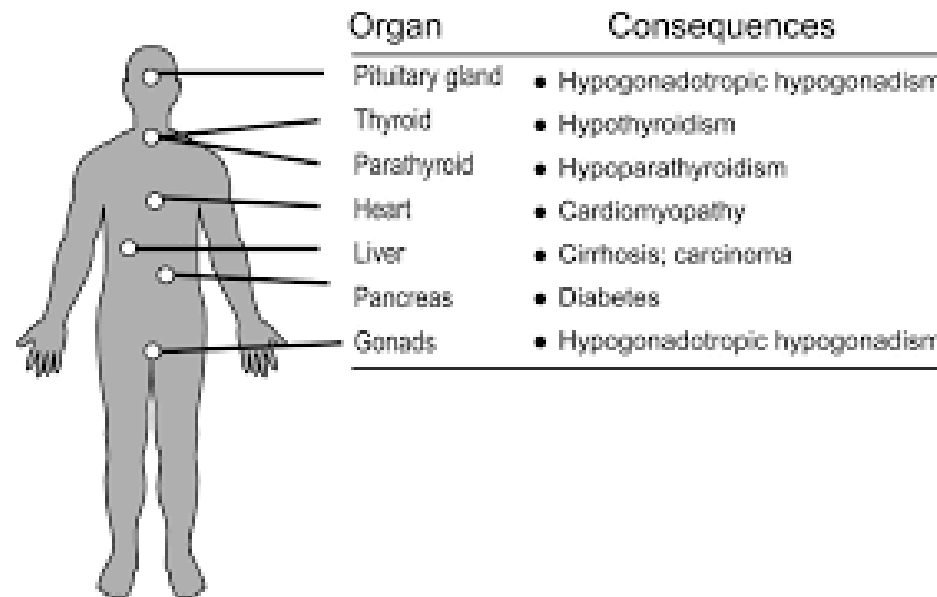
# Phase 3 IMerge: imetelstat vs supportive care in transfusion dependent MDS R/R or ineligible for ESAs

Measure, n (%)	Imetelstat N=118	Placebo N=60	<i>p</i> <sup>a</sup>
8-wk TI	47 (39.8)	9 (15.0)	<0.001
TI duration, median wks (95% CI) <sup>b</sup>	51.6 (26.9–83.9)	13.3 (8.0–24.9)	<0.001 <sup>d</sup>
24-wk TI	33 (28.0)	2 (3.3)	<0.001
HI-E <sup>c</sup>	50 (42.4)	8 (13.3)	<0.001

<sup>a</sup>Cochran Mantel Haenszel test stratified by prior transfusion burden and IPSS; <sup>b</sup>8-wk TI, Kaplan-Meier estimate; <sup>c</sup>2018 IWG; <sup>d</sup>Stratified log-rank test.

# Iron chelation

- Blood transfusions contain iron that can deposit over time in organs including heart and liver
- Increases risk of cardiac heart rhythm abnormalities and heart failure
- Elevation of liver enzymes



# BENEFIT OF IRON CHELATION THERAPY

Chelation can reverse some of the consequences of iron overload in MDS patients<sup>1,2</sup>

- By reducing serum ferritin and organ iron concentrations, it can potentially reduce morbidity
- 

Data from a registry of lower-risk Canadian MDS patients showed an improvement in median overall survival in patients who received ICT compared to non-chelated patients<sup>3</sup>

- This is supported by the results of additional studies that have shown a survival benefit of ICT in MDS patients<sup>4-7</sup>

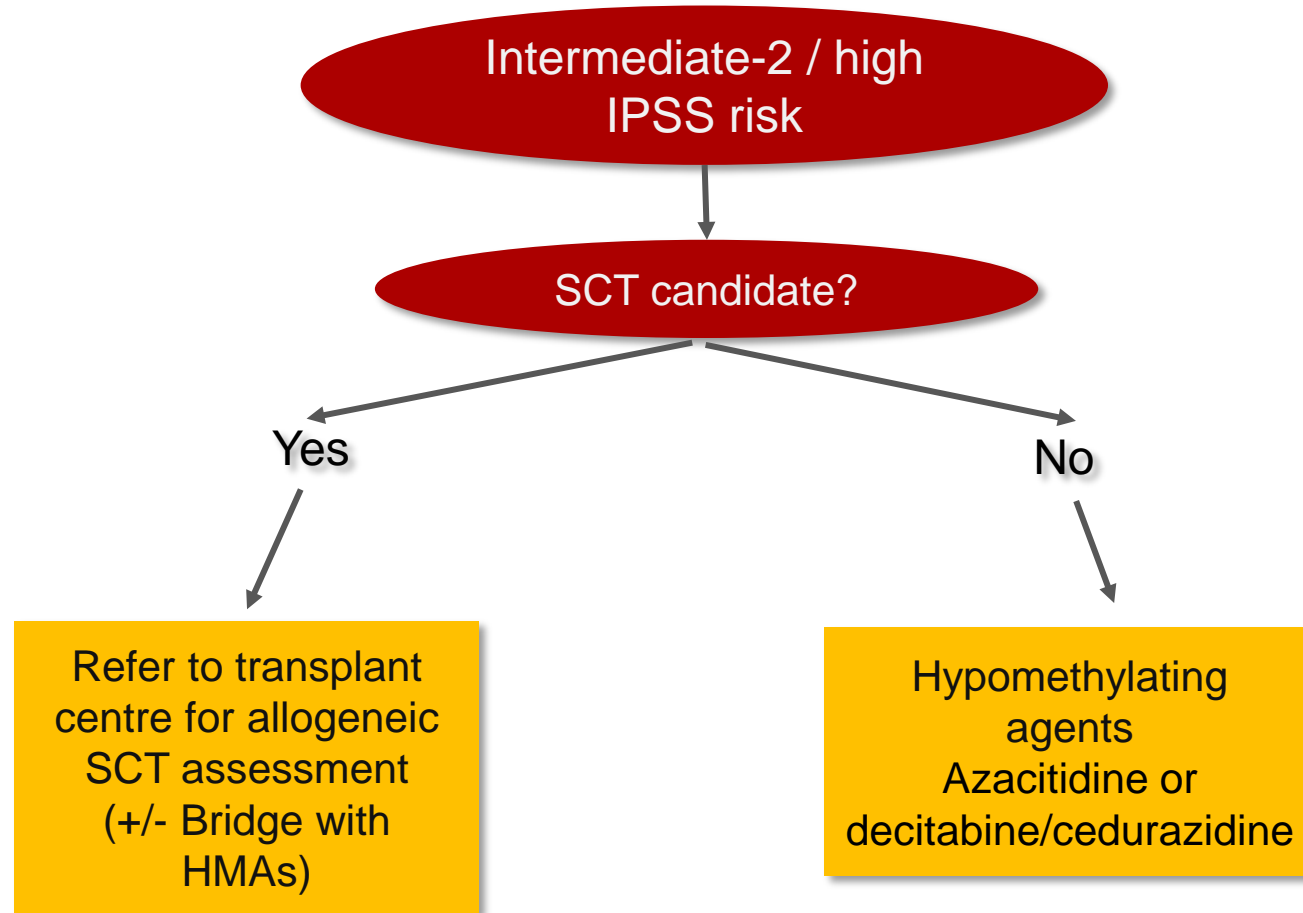
Further information on can be accessed here: <http://www.mdsironroad.org> (UN: ironroad; PW: makeirongreatagain) and here: <http://www.mdsclearpath.org>

ICT, iron chelation therapy; MDS, myelodysplastic syndromes; PW, password; UN, username

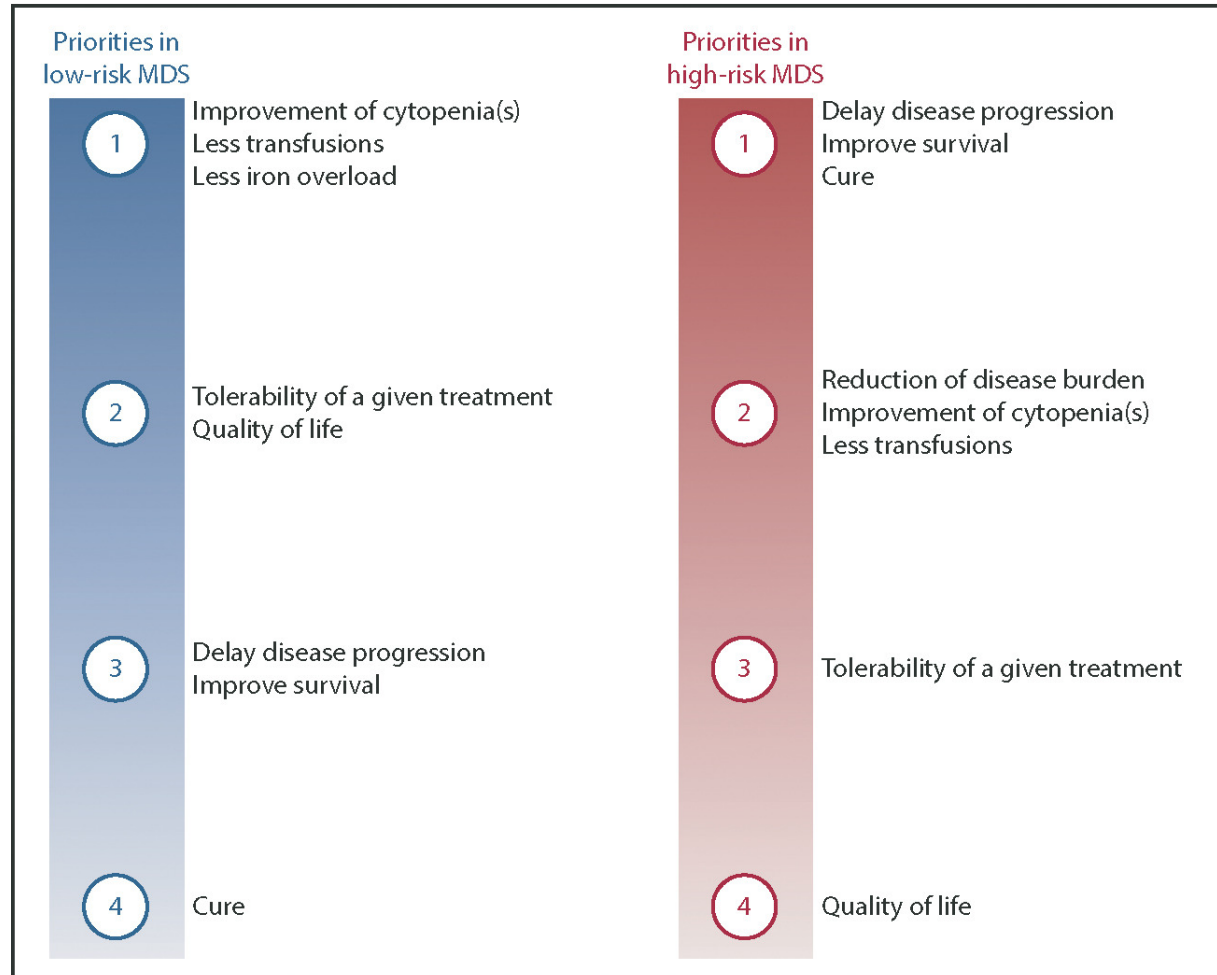
<sup>1</sup>Wells RA, et al. Leukemia Research. 2008;32:1338-1352. <sup>2</sup>Murray C, et al. Canadian Oncology Nursing Journal. 2016;26:19-28. <sup>3</sup>Leitch HA, et al. British Journal of Haematology. 2017;179:83-97.

<sup>4</sup>Neukirchen J, et al. Leukemia Research. 2012;36:1067-1070. <sup>5</sup>Leitch HA, et al. Clinical Leukemia. 2008;2:205-211. <sup>6</sup>Rose C, et al. Leukemia Research. 2010;34:864-870. <sup>7</sup>Raptis A, et al. Transfusion. 2010;50:190-199.

# Recommendations for patients with intermediate-2 / high IPSS risk



# Goals in care of patients with MDS



# Between a rock and a hard place

Transplantation is currently the only curative therapy for myelodysplastic syndromes

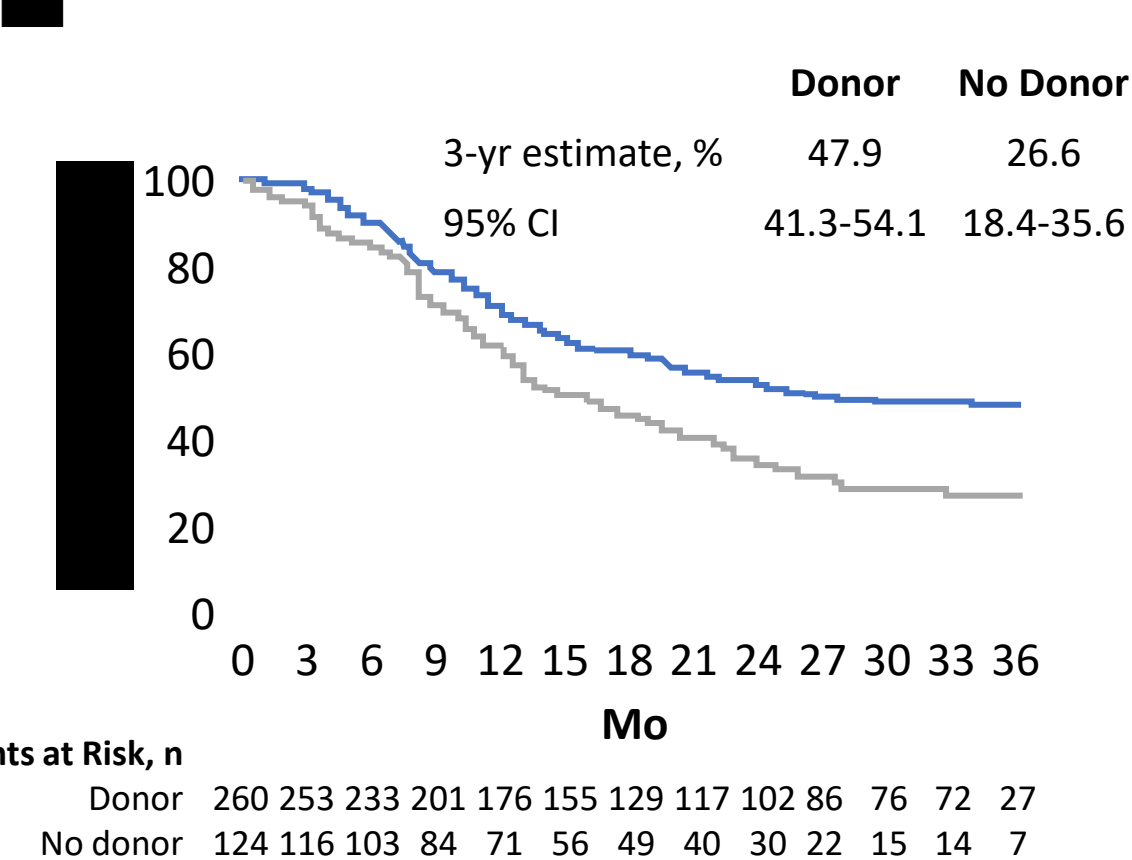


Median age at diagnosis is 65-70  
Toxicity of transplantation can be prohibitive

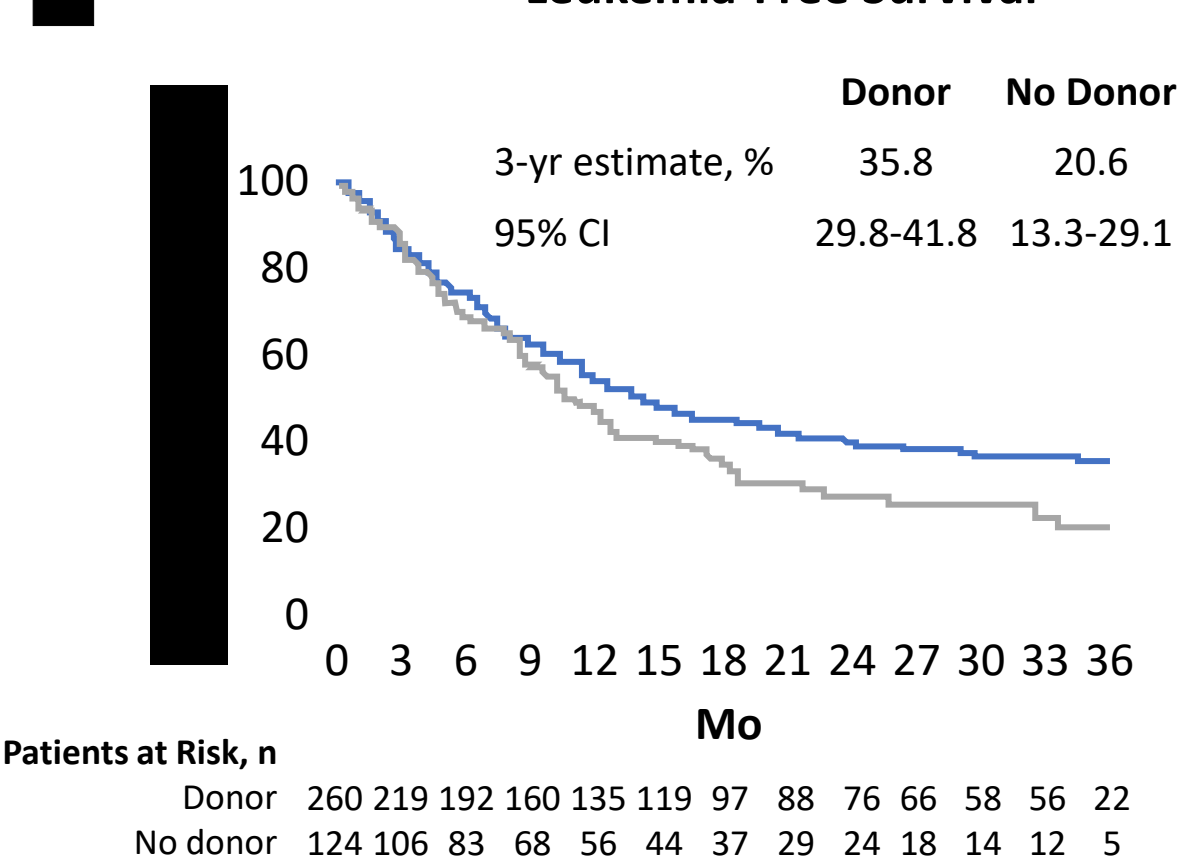


# BMT CTN 1102: Reduced intensity conditioning plus Allotransplant vs Best nontransplant care in Older Patients (50-75y) With Higher-Risk MDS

Overall Survival

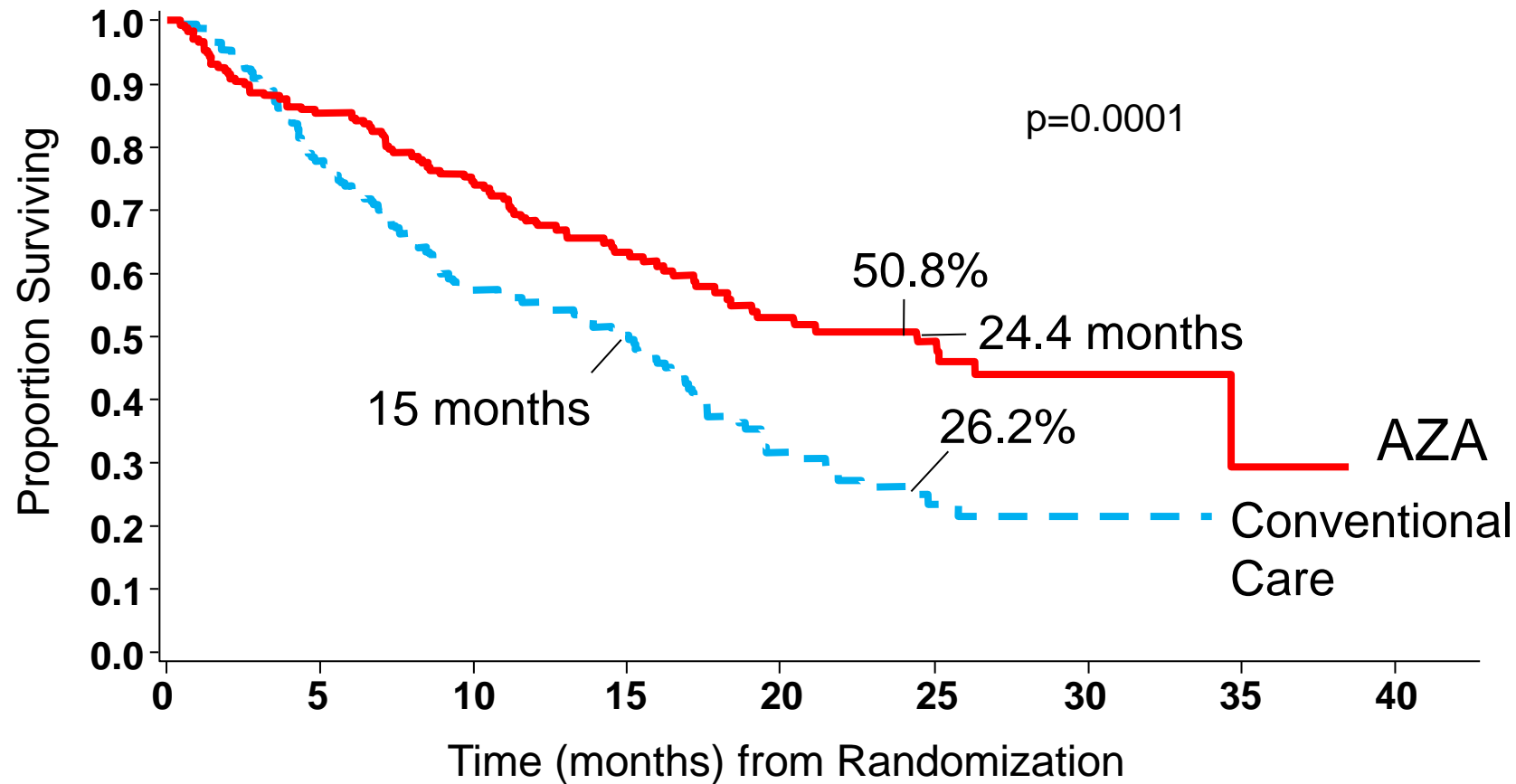


Leukemia-Free Survival



Biological randomization, N=384

# Overall Survival: Azacitidine vs Conventional Care Regimen – AZA001

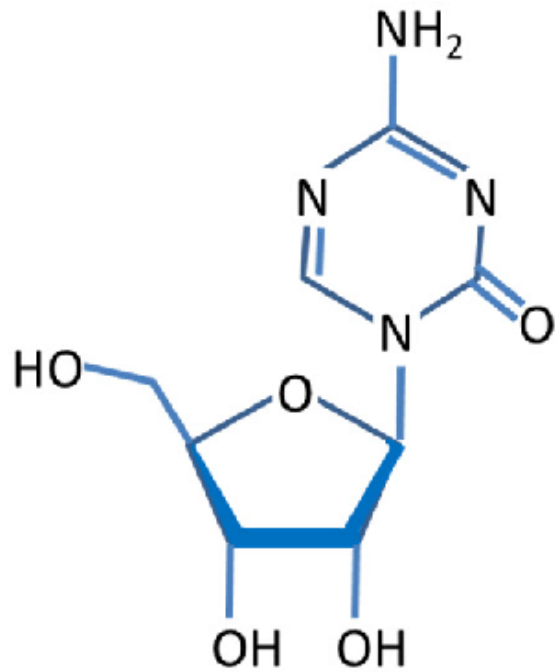


Survival Difference 9.4 months

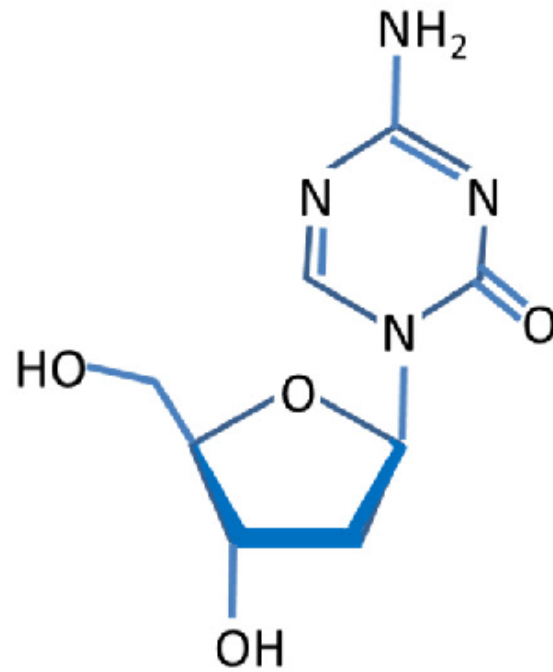
HR = 0.58 [95% CI: 0.43, 0.77]

Deaths: AZA = 82, Conventional Care = 113

# Decitabine and azacytidine are hypomethylating agents



azacytidine



decitabine

- Azacytidine is metabolized intracellular into decitabine
- Azacytidine is administered SC or IV with short half life
  - peak plasma concentration at 41 minutes after SC administration
- Decitabine is administered IV with similar half life 37-47 min
- Oral version with cedurazidine

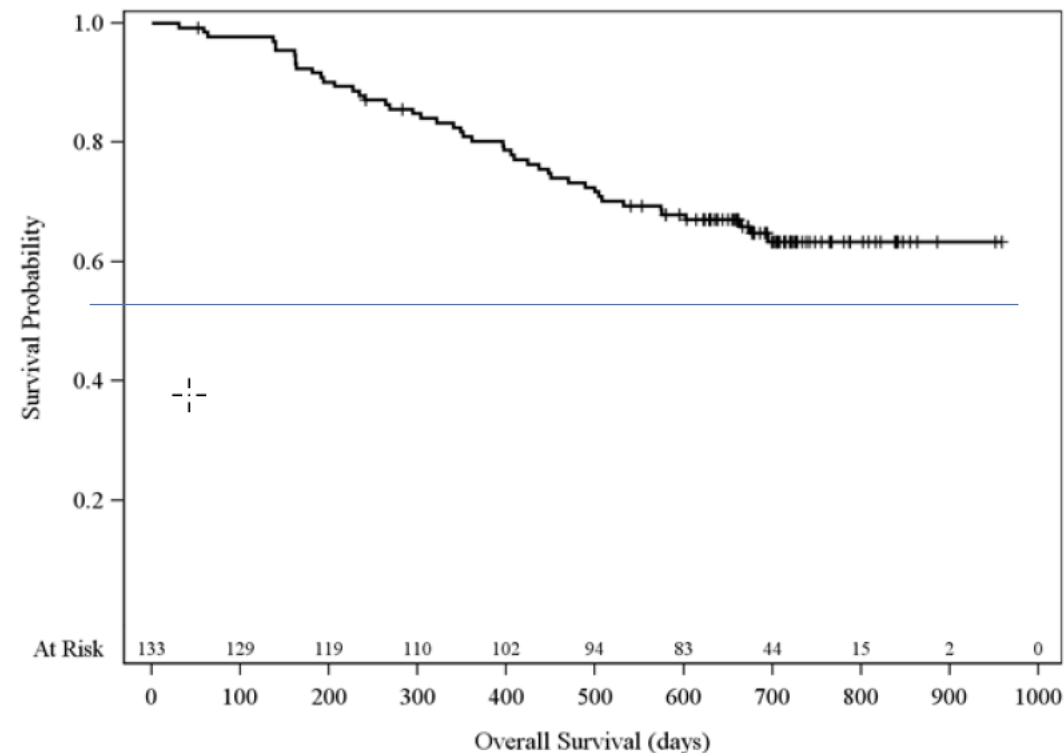
# Phase - 3 Results for oral decitabine cedurazidine

## Phase 3 Response rates:

Efficacy Endpoints	Phase 3 Overall (N=133)	
	n (%)	95% CI
Complete Response (CR)	29 (22)	(15.1, 29.8)
Partial response (PR)	0	
Marrow CR (mCR)	43 (32.3)	
Marrow CR (mCR) with Hematologic Improvement	22 (16.5)	
Hematological Improvement (HI) %	10 (7.5)	
<b>Overall Response (OR = CR + PR + mCR + HI) n (%)</b>	<b>82 (61.7)</b>	<b>(52.8, 69.9)</b>
RBC transfusion independence n (%)	27/53 (51)	
Platelet transfusion independence n (%)	6/12 (50)	
Median CR duration	14.0 months	
Median duration of best response	12.7 months	

**34 (26%) of the 133 patients went on to stem cell transplantation following treatment.**

## Phase 3 Overall Survival (OS):



- Median follow up is 24.7 months
- mOS has not yet been reached
- Median Leukemia Free survival is 33 months<sup>2</sup>

# Oral decitabine/cedazuridine (DEC/C)

- Fixed dose oral tablet approved for MDS with excess blasts, CMML, Int-1 and Int-2 or high risk IPSS subtypes
- Patients may be initiated on either hypomethylating agent (azacitidine or DEC/C)
- Initiate at 5 tabs/28 days and continue until maximal response
  - Median marrow response at 2.2 months
  - Suggest bone marrow aspirate and biopsy at 4 months to assess disease response and marrow cellularity. May need dose delay/ dose reduction if cytopenias in the context of marrow response.

# Switching from subcutaneous azacitidine to oral decitabine cedurazidine

- If switching patients in remission from parenteral azacitidine to oral DEC/C, we consider dose reduction to minimize low blood counts especially in the following patient populations:
  - Older or other medical conditions
  - Patients with low blood counts already on azacitidine (ie neutrophils  $<1 \times 10^9$  or platelets  $<30$ )
  - Patients who have already needed dose delays or dose reductions to azacitidine (ie every 5 or 6 weeks) for low blood counts
  - Less cellular marrow due to long term therapy with azacitidine or underlying disease

# Unmet needs in care of patients in Canada

- Management of overlap syndromes (myelodysplastic syndromes and myeloproliferative neoplasms)
- Options for treatment of relapsed MDS
- Targeted therapy for TP53 mutated MDS
- Improving outcomes for MDS
  - Phase 3 trial imetelstat for lower risk MDS
  - Phase 3 VERONA trial with azacitidine and venetoclax (14d) for MDS
- Many others in pipeline

Clinical trials are investigating new types of treatments





# Other supports available

- Home care
- Wellspring
- AAMAC
- Leukemia and lymphoma society
- Psychosocial services
- Dieticians
- Social work, home care, mobile lab

# Thank you!



PATIENT SUPPORT  
GROUP MEETING



RÉUNION DU GROUPE DE  
SOUTIEN AUX PATIENTS

Our patients are why we are here  
Caregivers are a huge part of treatment success  
Registry has been increasing knowledge of disease  
Clinical trials are teaching us more

# Questions?

