Therapie älterer Patienten mit AML

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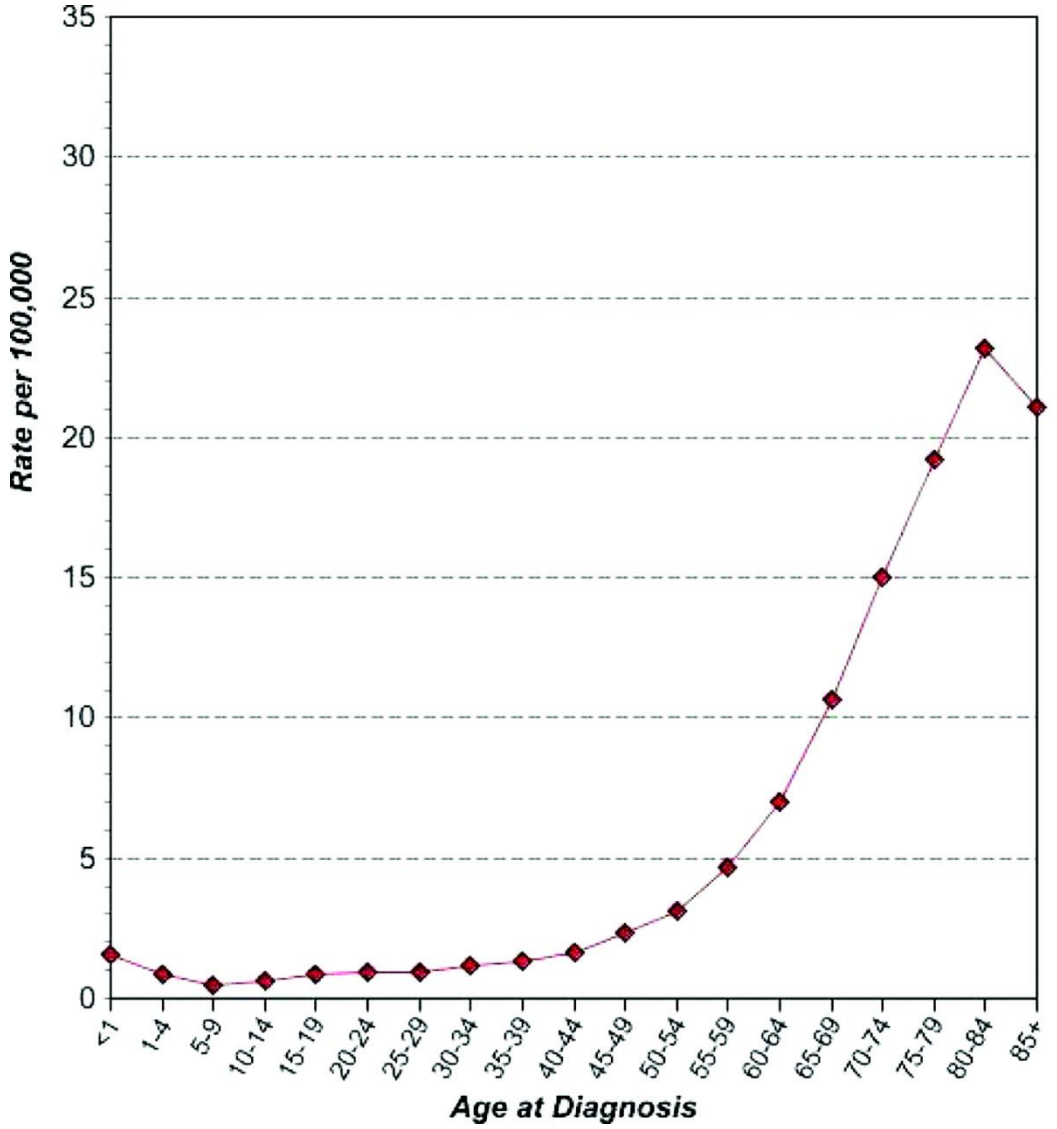
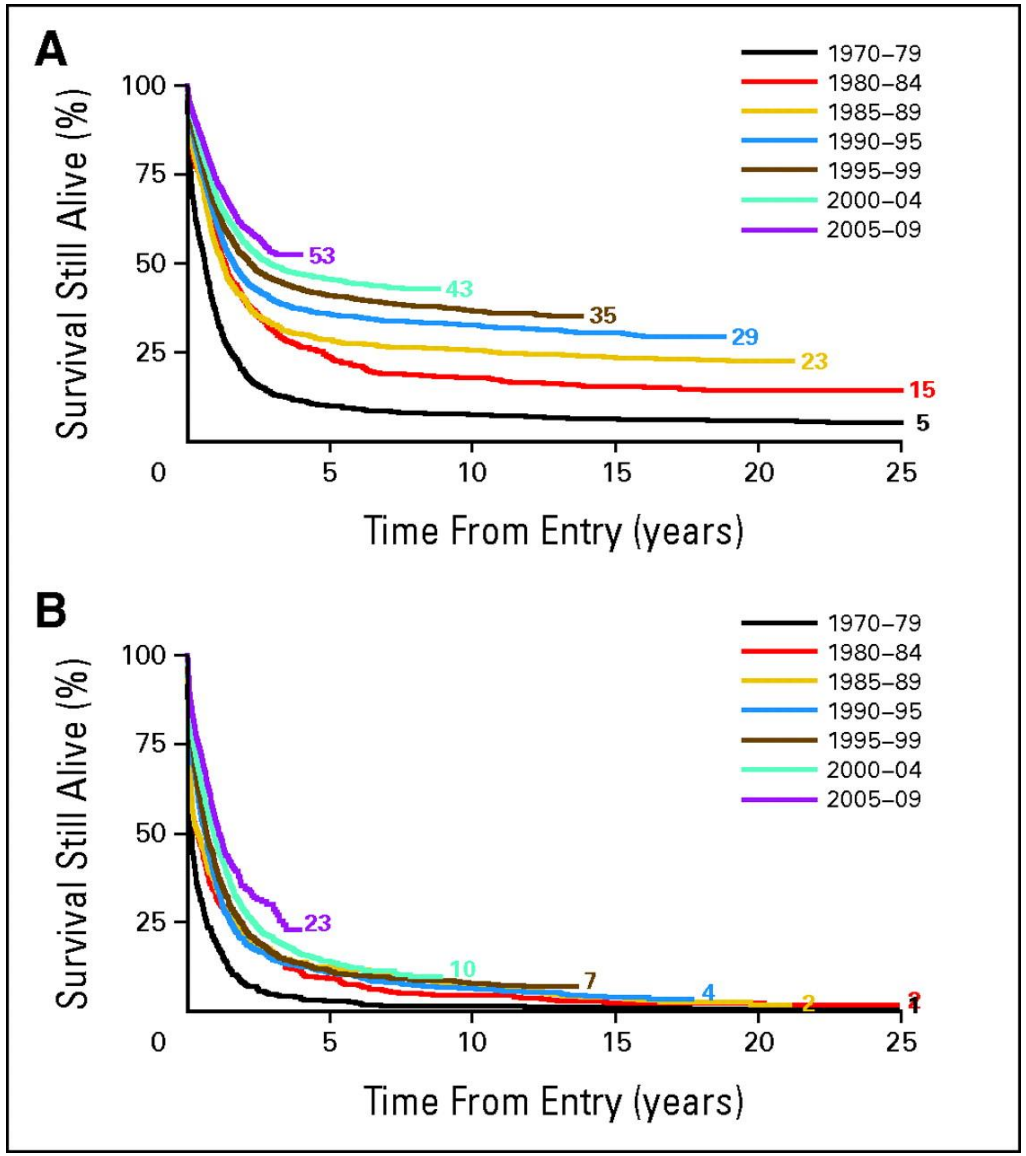
The incidence of AML as a function of age; 2000–2005 Surveillance Epidemiology and End Results (SEER) Data



Klepin H D , and Balducci L The Oncologist 2009;14:222-232

Change in overall survival with time.



Burnett A et al. JCO 2011;29:487-494

Unfavorable tumor biology is more frequent in the elderly.

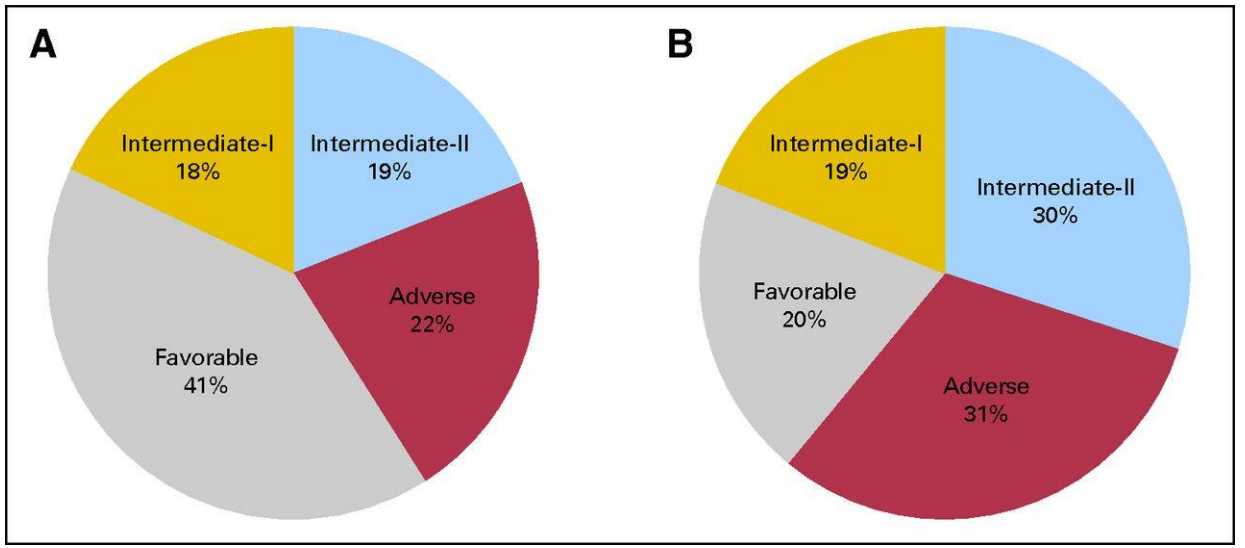
Table 1. Unfavorable tumor biology is more frequent in the elderly

Biologic characteristic	Examples	Proportion of elderly AML affected	References
Unfavorable cytogenetic abnormalities	Chromosome 5 or 7 abnormality Complex karyotype	22%–50%	[2, 5, 9, 13, 22]
Multidrug resistance phenotype	MDR1 overexpression	58%–71%	[13]
Preceding hematologic disease	Myelodysplastic syndrome	21%–34%	[21, 22]

Abbreviations: AML, acute myelogenous leukemia; MDR, multidrug resistance.

Klepin H D , and Balducci L The Oncologist 2009;14:222-232

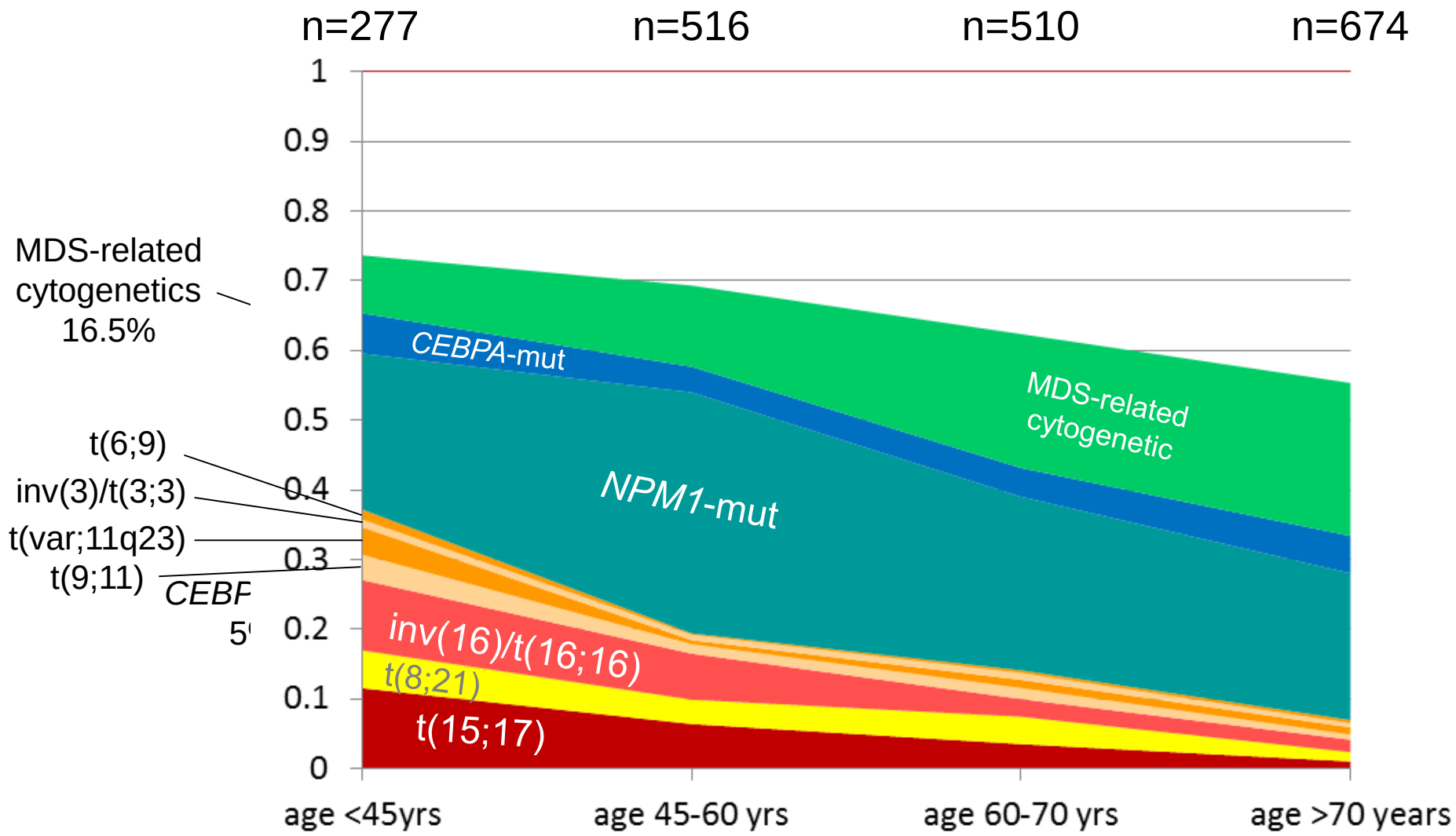
Distribution of the European LeukemiaNet genetic groups in younger (A) and older (B) adults with primary acute myeloid leukemia.



Mrózek K et al. JCO 2012;30:4515-4523

Distribution of Genetically Defined WHO Categories Based on Fast Biomarker Screening

AMLSG-BiO 2011-2013 [NCT01252485, n=1977; median age 65 years (18-92)]



Auswahl älterer Patienten für die Intensive Chemotherapie

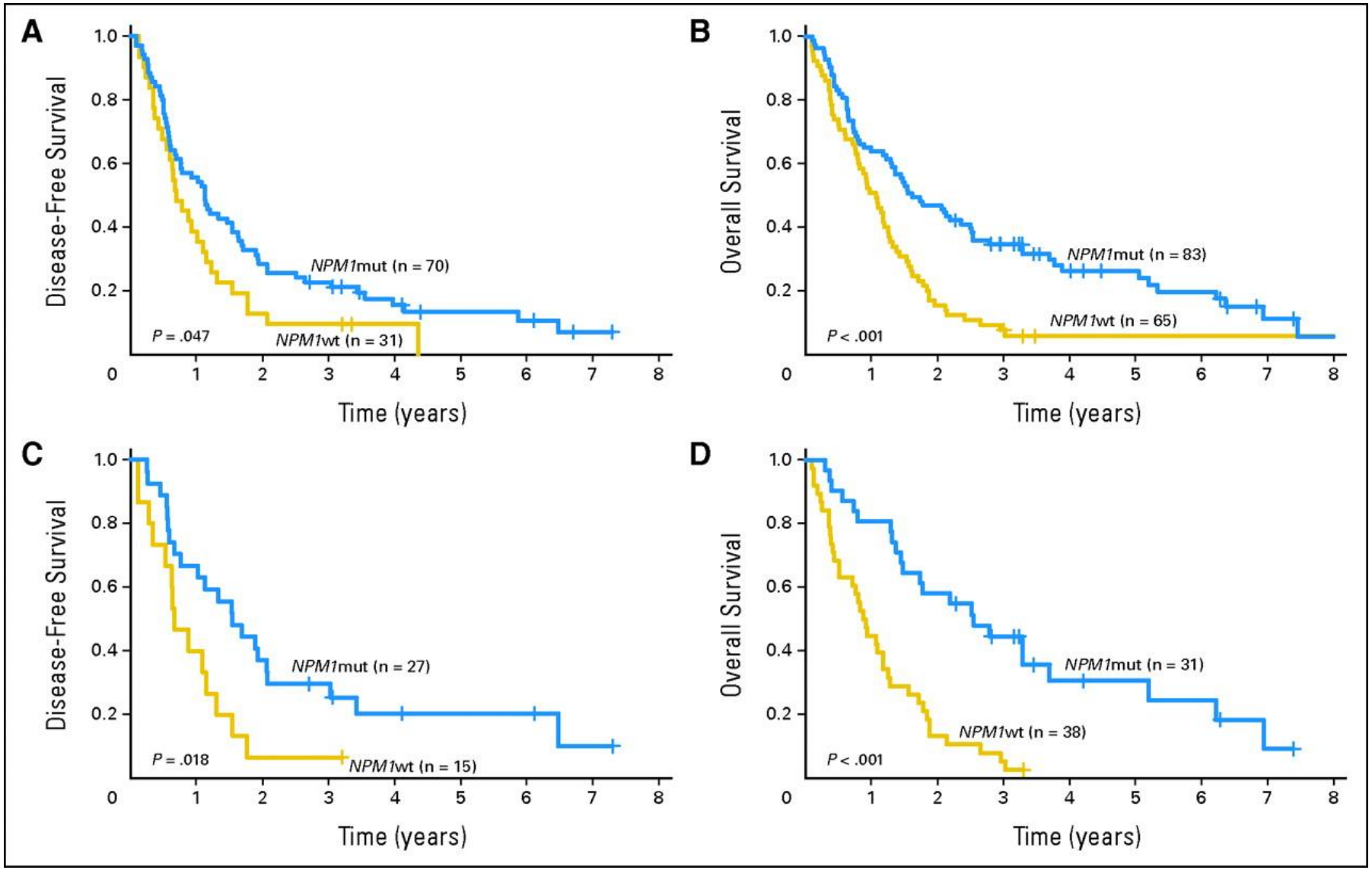
- „Biologisches“ Alter
- Comorbiditäten
- Organfunktionen
- Karyotyp
- Molekulare Veränderungen
- Scores

Response-Raten nach „7+3“ Chemotherapie bei Patienten mit AML über 60 Jahre mit normalem Karyotyp

	NPM-1 wt	NPM-1 mut	p
Alter 60-69 Jahre	N=27	N=52	
CR Rate	59%	83%	0.031
Alter ≥ 70 Jahre	N=38	N=31	
CR Rate	39%	87%	<0.01

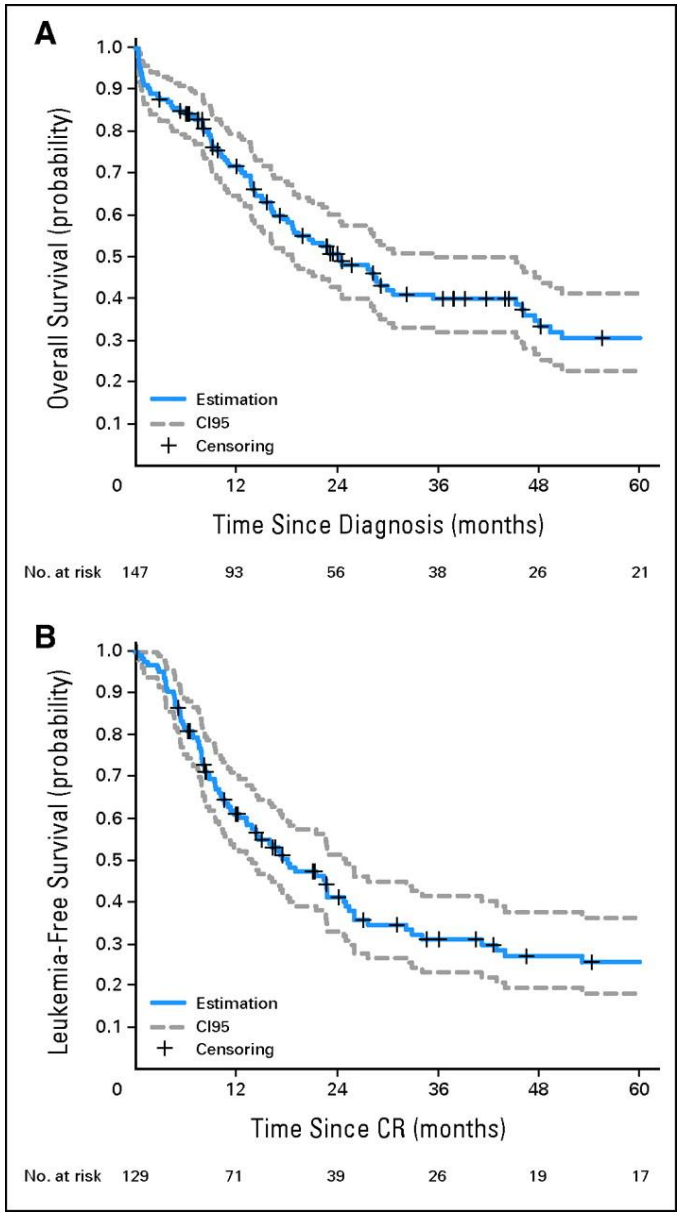
Becker H et al. JCO 2010;28:596-604

(A) Disease-free survival and (B) overall survival of patients age ≥ 60 years with cytogenetically normal de novo acute myeloid leukemia according to NPM1 mutation status



Becker H et al. JCO 2010;28:596-604

Outcome of Patients with CBF Leukemias over an age of 60 Years



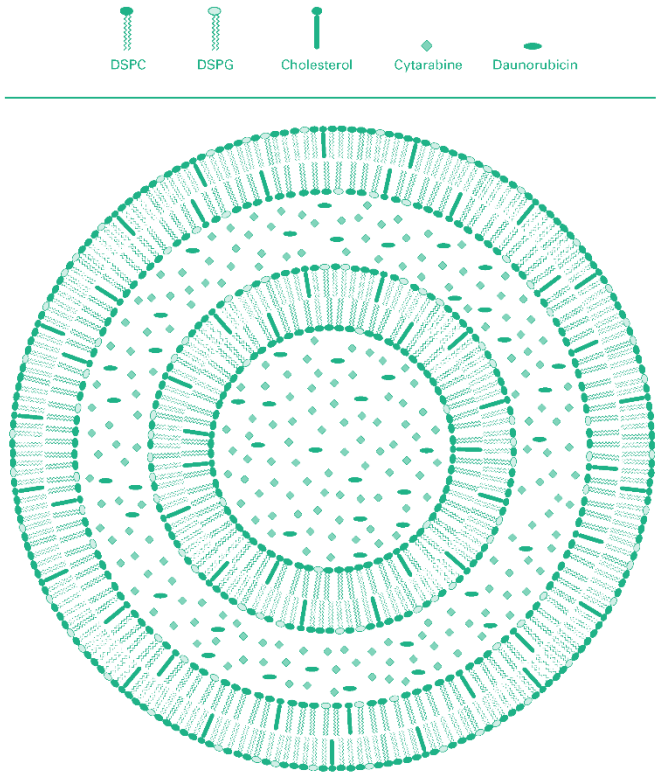
Prébet T et al. JCO 2009;27:4747-4753

Intensive Therapie für ältere Patienten mit Hochrisikomerkmalen

CPX-351

CPX-351 is a liposomal formulation of cytarabine and daunorubicin encapsulated at a 5:1 molar ratio

- Fixed molar ratio maintained in human plasma for at least 24 hours after final dose[1]
- Drug exposure maintained for 7 days[1]
- Selective uptake by leukemic vs normal cells in bone marrow of leukemia-bearing mice[2]

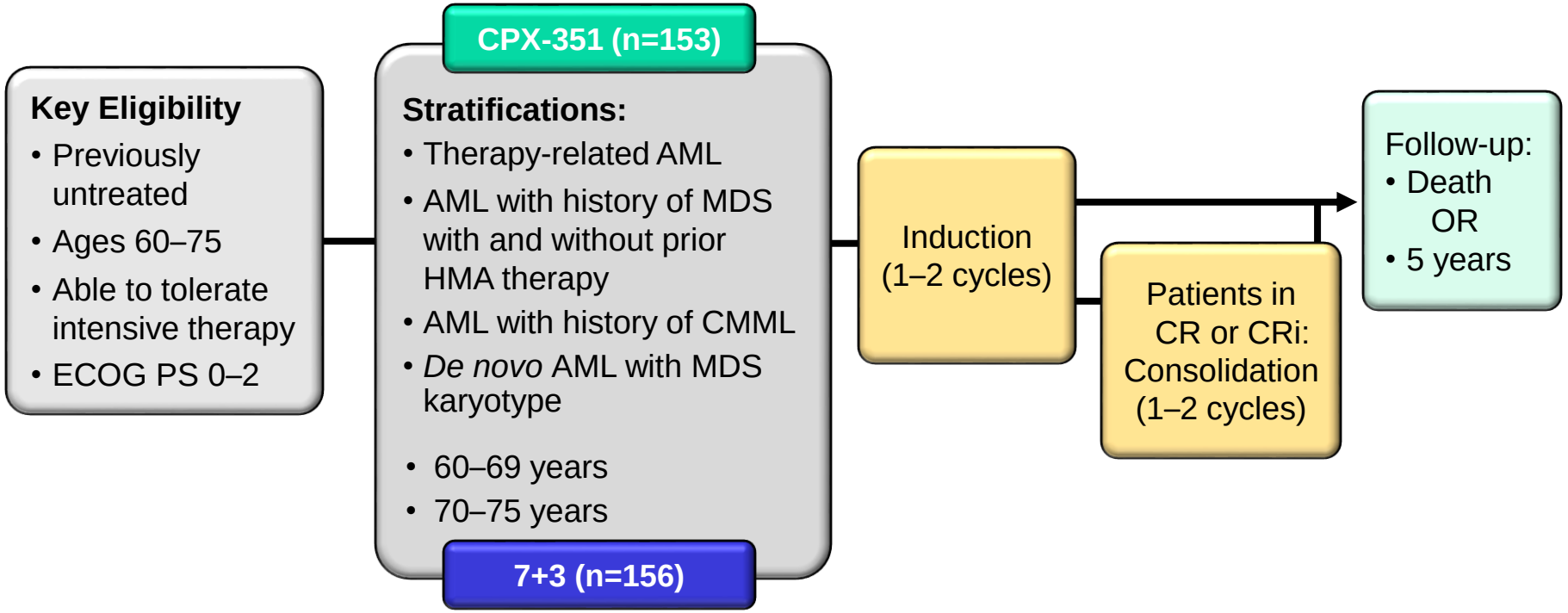


1. Feldman EJ et al. *J Clin Oncol.* 2011;29(8):979–985; 2. Lim WS et al. *Leuk Res.* 2010;34(9):1245–1223.

CPX-351 Phase III Study Design

Randomized, open-label, parallel-arm, standard therapy–controlled

1:1 randomization

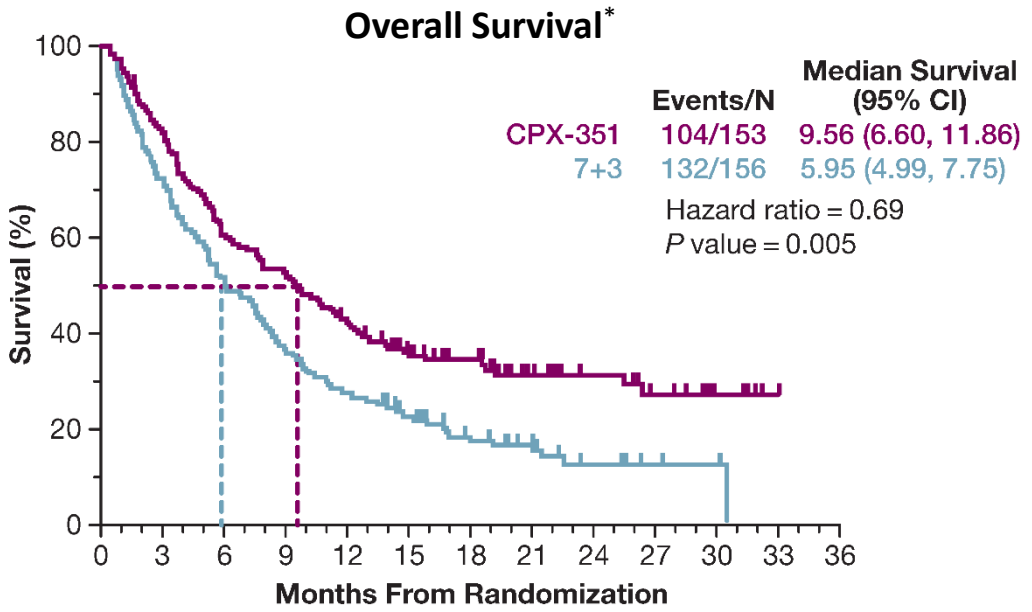


AML, acute myeloid leukemia; CMML, chronic myelomonocytic leukemia; CR, complete response; CRi, CR with incomplete platelet/neutrophil recovery; ECOG PS, Eastern Cooperative Oncology Group performance status; HMA, hypomethylating agents; MDS, myelodysplastic syndrome.

1. World Health Organization. *WHO Classification of Tumours of Haematopoitic and Lymphoid Tissues*. Swerdlow S et al (ed). Lyon, IRAC Press, 2008.

Clinical Results of Phase 3 Study

	CPX-351 (n=153)	7+3 (n=156)		
	Median Survival in Months (95% CI)		Hazard Ratio	*P* value
Event-Free Survival	2.53 (2.07, 4.99)	1.31 (1.08, 1.64)	0.74 (0.58, 0.96)	0.021
Remission Duration	6.93 (4.60, 9.23)	6.11 (3.45, 8.71)	0.77 (0.47, 1.26)	0.291
Deaths ≤ 60 Days*	13.8%	21.8%		
			Odds Ratio	*P* value
CR+CRi	47.7%	33.3%	1.77 (1.11, 2.81)	0.016
HCT	34.0%	25.0%	1.54 (0.92, 2.56)	0.098

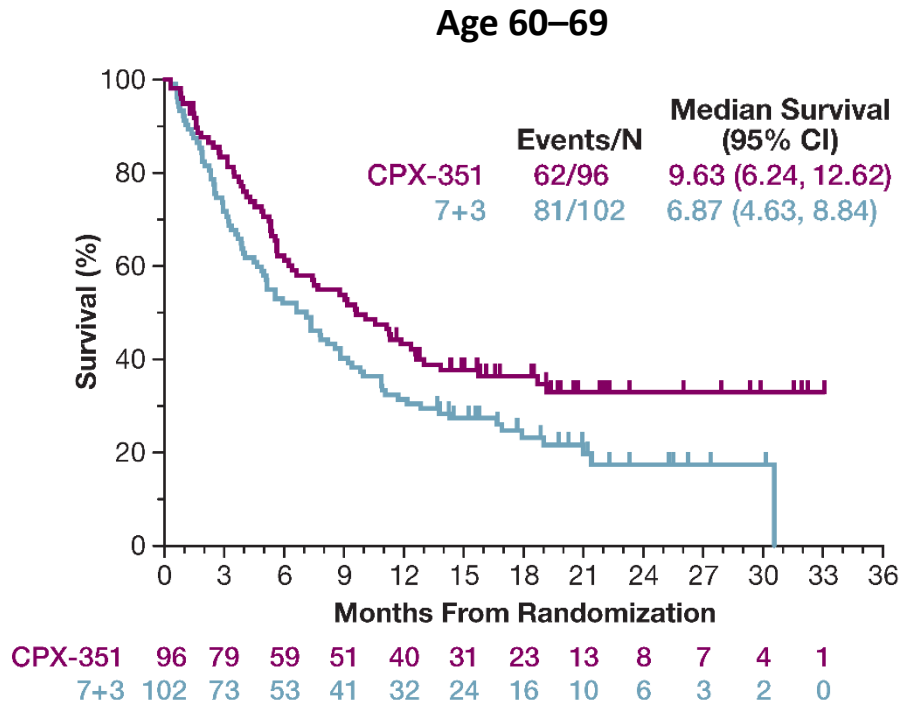


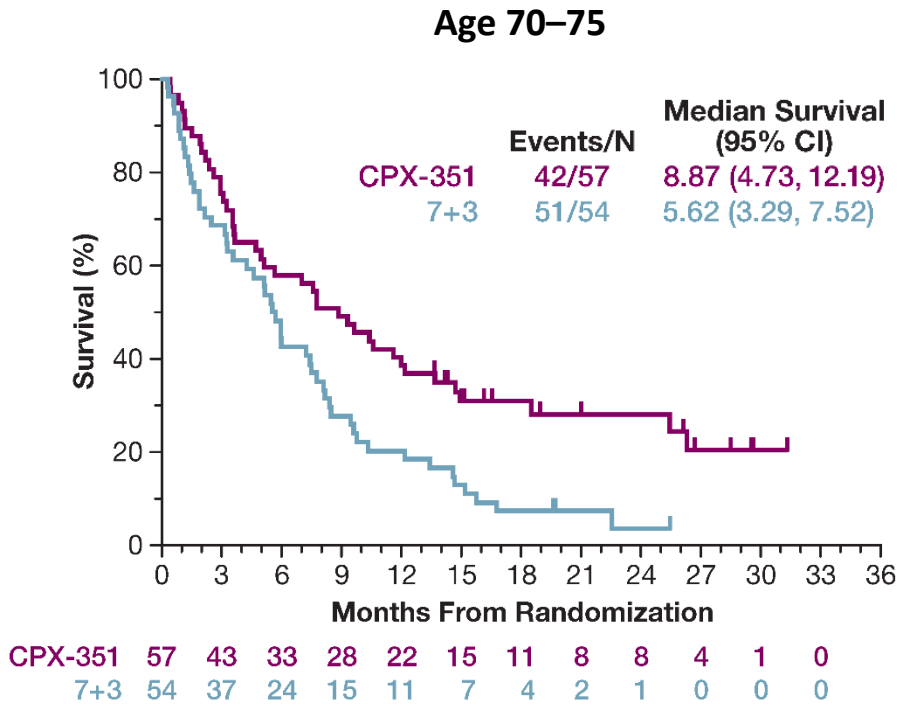
*Kaplan-Meier estimate.

CI, confidence interval; CR, complete response; CRi, CR with incomplete platelet/neutrophil recovery; HCT, Hematopoietic Cell Transplant.

Exploratory Analysis by Age: Overall Survival

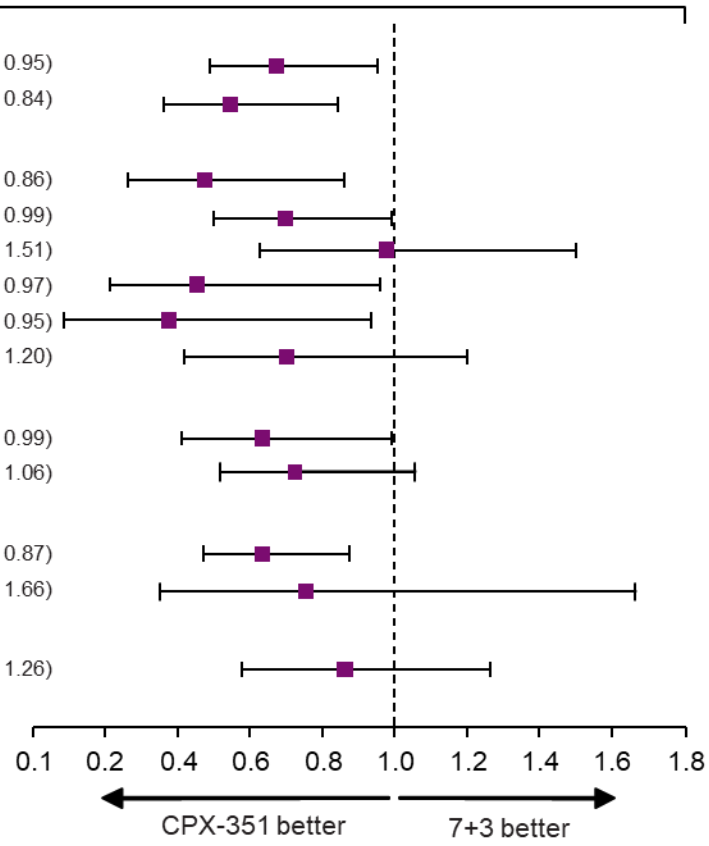
- Age 60–69 years, hazard ratio of 0.68 (95% CI: 0.49, 0.95)
- Age 70–75 years, hazard ratio of 0.55 (95% CI: 0.36, 0.84)





Überlebensvorteile auch in weiteren Subgruppen sichtbar

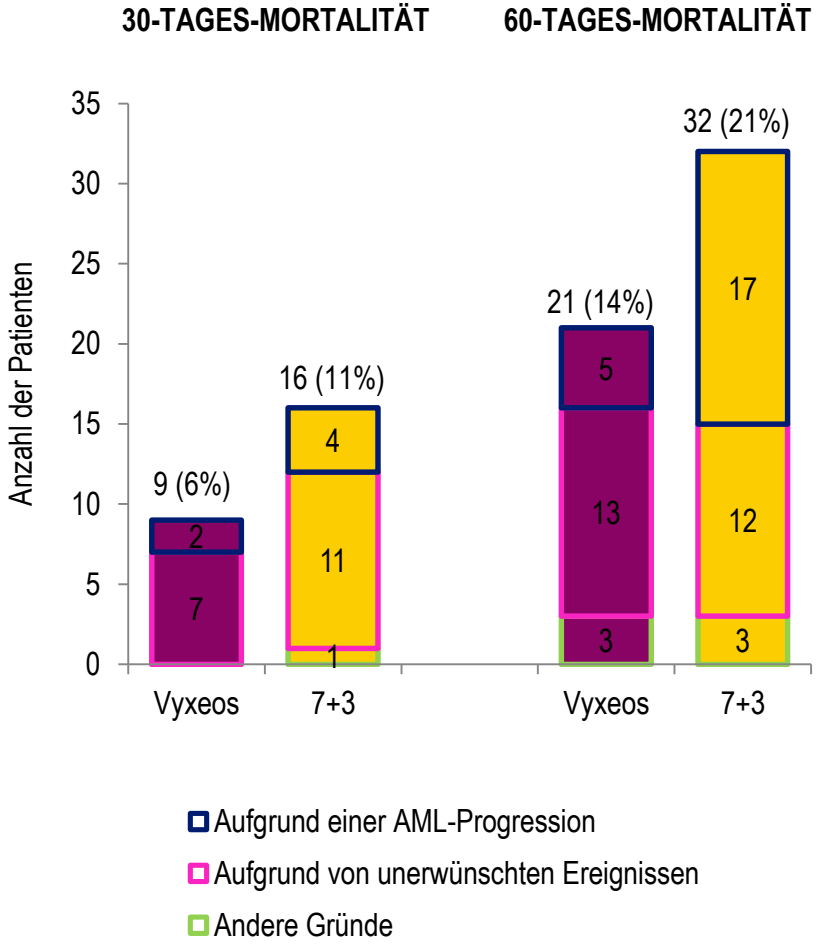
Subgroup	CPX-351 n	CPX-351 Median OS, mo	7+3 n	7+3 Median OS, mo	Hazard ratio (95% CI) for death
Age					
60-69 years	96	9.63	102	6.87	0.68 (0.49, 0.95)
70-75 years	57	8.87	54	5.62	0.55 (0.36, 0.84)
Type of AML					
Therapy-related AML	30	12.17	33	5.95	0.48 (0.26, 0.86)
AML with antecedent MDS or CMML	82	7.38	86	5.95	0.70 (0.50, 0.99)
MDS with prior HMA exposure	50	5.65	55	7.43	0.98 (0.64, 1.51)
MDS without prior HMA exposure	21	15.74	19	5.13	0.46 (0.21, 0.97)
CMML	11	9.33	12	2.28	0.37 (0.14, 0.95)
de novo AML with MDS karyotype	41	10.09	37	7.36	0.71 (0.42, 1.20)
Cytogenetic risk at screening					
Favorable/intermediate	71	14.72	63	8.41	0.64 (0.41, 0.99)
Unfavorable	72	6.60	83	5.16	0.73 (0.51, 1.06)
Baseline FLT3 mutation status					
FLT3 wild type	116	9.33	120	5.98	0.64 (0.47, 0.87)
FLT3 mutation	22	10.25	21	4.60	0.76 (0.34, 1.66)
Overall HMA experience					
All patients with prior HMA exposure[a]	62	5.65	71	5.90	0.86 (0.59, 1.26)



Lancet JE *et al*. 2018. J Clin Oncol 2018.

Verlängerte Phase der Neutropenie führt nicht zur Erhöhung der Therapie-bedingten Mortalität

	ANC ≥ 500/uL		Thrombozyten ≥ 50,000/uL	
	Vyxeos	7 + 3	Vyxeos	7 + 3
Nach der 1. Induktion	n=58	n=34	n=58	n=34
Median (Tage)	**35**	**29**	**36.5**	**29**
Nach der 2. Induktion	n=15	n=18	n=15	n=18
Median (Tage)	**35**	**28**	**35**	**24**



Lancet JE, et al. *J Clin Oncol* 2016;34(suppl):7000
Kolitz JE, et al. Poster presented at ASCO Annual Meeting 2017; Abstract 7036.

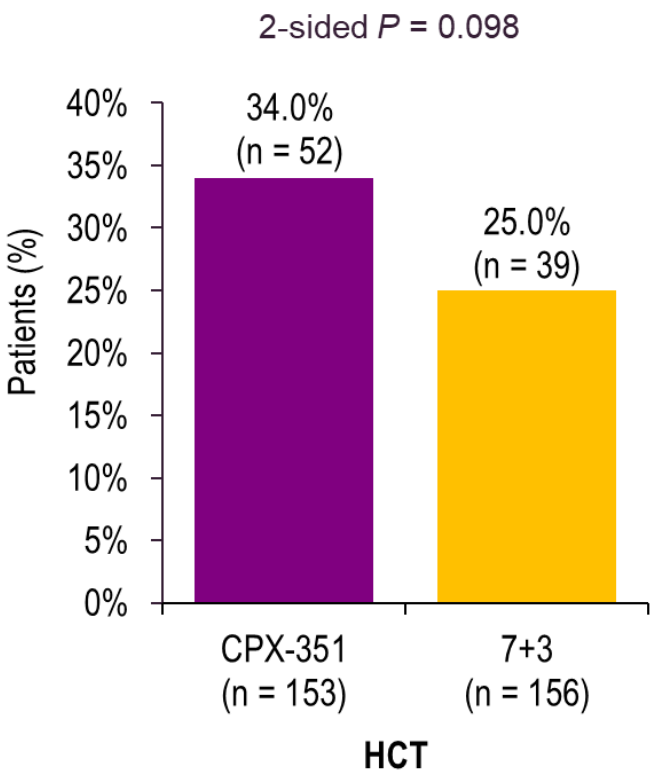
Safety Profile

- Grade 3–4 AEs and AEs resulting in death were generally similar between arms
  - Differences in infection and bleeding events were associated with delayed recovery from myelosuppression in the CPX-351 arm

Events MedDRA Preferred Term	60–69		70–75	
	CPX-351 n=96 n (%)	7+3 n=102 n (%)	CPX-351 n=57 n (%)	7+3 n=54 n (%)
Any grade 3–4 AE	85 (89)	91 (93)	50 (88)	45 (85)
Any serious AE*	49 (51)	36 (37)	22 (39)	22 (42)
Febrile neutropenia	7 (7.3)	5 (5.1)	4 (7.0)	3 (5.7)
Sepsis	6 (6.3)	2 (2.0)	2 (3.5)	2 (3.8)
Respiratory failure	4 (4.2)	7 (7.1)	3 (5.3)	1 (1.9)
Acute respiratory failure	4 (4.2)	1 (1.0)	1 (1.8)	2 (3.8)
Ejection fraction decreased	3 (3.1)	5 (5.1)	3 (5.3)	1 (1.9)
Pneumonia	3 (3.1)	3 (3.1)	3 (5.3)	1 (1.9)
Disease progression	1 (1.0)	3 (3.1)	1 (1.8)	1 (1.9)
Hypoxia	1 (1.0)	3 (3.1)	1 (1.8)	0
Pulmonary edema	0	1 (1.0)	1 (1.8)	2 (3.8)
Any AE resulting in death	8 (8.3)	11 (11)	6 (11)	11 (21)

*Specific serious AEs occurring in ≥2% of patients in either age group are listed. MedDRA, Medical Dictionary for Regulatory Activities.

Anteil der Transplantierten konnte unter CPX-351 deutlich erhöht werden, besonders bei den über 70-Jährigen



HSCT Baseline Characteristics		CPX-351 n (%)	7+3 n (%)
Patients who went to transplant		52 (34)	39 (25)
Age	60–69	36 (70)	33 (85)
	70–75	16 (31)	6 (15)
PS	0–1	48 (92)	37 (95)
	2	4 (8)	2 (5)
Karyotype	Intermediate	27 (52)	18 (46)
	Poor	21 (40)	19 (49)
	Unknown	4 (8)	2 (5)
Strata	tAML	11 (21)	9 (23)
	MDS with prior HMA	14 (27)	14 (36)
	MDS without prior HMA	7 (14)	5 (13)
	CMML	3 (6)	0 (0.0)
	de novo	17 (33)	11 (28)
Transplanted in CR/CRi		39 (75)	24 (62)
Transplanted in NR (no response)		8 (15)	3 (8)

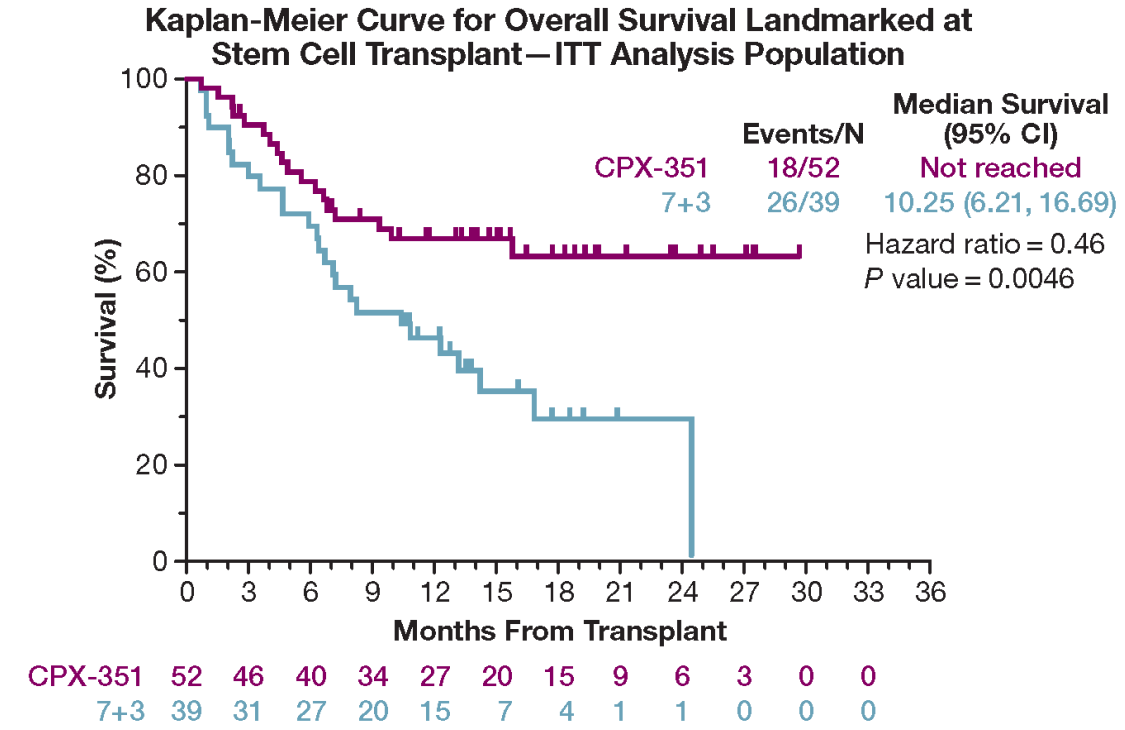
Lancet JE *et al*. 2018. J Clin Oncol 2018.

Survival Landmarked from Time of Transplant

- CPX-351 median OS not reached vs 10.25 months for 7+3

  HR of 0.46 favoring CPX-351 (*P*=0.0046)

  Cox proportional hazards HR, including transplant as a time-dependent covariate, was 0.51 (95% CI, 0.35–0.75; *P*=0.0007), favoring CPX-351



CI, confidence interval; HR, hazard ratio; OS, overall survival.

Lancet JE ASH 2016

Venetoclax with low-dose cytarabine induces rapid, deep, and durable responses in previously untreated older adults with AML ineligible for intensive chemotherapy

Andrew H. Wei[1], **Stephen Strickland**[2], Jing-Zhou Hou[3], Walter Fiedler[4], Tara L. Lin[5], Roland B. Walter[6], Anoop Enjeti[7], Wan-Jen Hong[8], Brenda Chyla[9], Qin Qin[9], Relja Popovic[9], Kaffa Fakouhi[9], John Hayslip[9], Gail J. Roboz[10]

[1]The Alfred Hospital and Monash University, Melbourne, VIC, Australia; [2]Vanderbilt-Ingram Cancer Center, Nashville, TN, USA; [3]University of Pittsburgh Medical Center Cancer Center, Pittsburgh, PA, USA; [4]Hubertus Wald University Cancer Center, University Medical Center Hamburg-Eppendorf, Hamburg, Germany; [5]University of Kansas Medical Center, Kansas City, KS, USA; [6]Clinical Research Division, Fred Hutchinson Cancer Research Center, Seattle, WA; [7]Calvary Mater Hospital Newcastle, Waratah, NSW, and School of Medicine and Public Health, University of Newcastle, Callaghan, NSW, Australia; [8]Genentech, Inc., South San Francisco, CA, USA; [9]AbbVie, Inc., North Chicago, IL, USA; [10]Weill Cornell Medical College, New York, NY, USA

Enrollment Criteria

KEY INCLUSION CRITERIA	KEY EXCLUSION CRITERIA
AML by histological confirmation	Prior treatment for AML, except hydroxyurea
Age ≥60 years	Prior treatment with HMA for preexisting myeloid disorder
Ineligible for standard induction therapy with cytarabine and anthracycline	Active CNS involvement
ECOG score 0–2 for patients ≥75 years	WBC count >25 $\times 10^9$ per liter
ECOG score 0–3 for patients 60-74 years	Infection with HIV, HBV, or HCV
Adequate renal and hepatic function	

Abbreviations: CNS, central nervous system; ECOG, European Collaborative Oncology Group; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus; NCCN, National Comprehensive Cancer Network; WBC, white blood cell

Patient Characteristics

Characteristic	N=82
Median age (range), years	74 (63–90)
Male, n (%)	53 (65)
ECOG Performance Score, n (%)	
0	12 (15)
1	46 (56)
2	23 (28)
3	1 (1)
Baseline bone marrow blasts, n (%)	
<30%	27 (33)
≥30 – <50%	18 (22)
≥50%	36 (44)
Secondary AML, n (%)	40 (49)
Prior HMA treatment, n (%)	24 (29)
CYP3A inhibitor use, n (%)	41 (50)

AML, acute myeloid leukemia; ECOG, Eastern Cooperative Oncology Group; HMA, hypomethylating agent

Cytogenetics and Mutations	N=82*
Cytogenetics, n (%)*	
Intermediate risk	49 (60)
Poor risk	26 (32)
No mitosis	7 (8)
Somatic mutations, n (%)†	
TP53	10 (14)
FLT3	16 (23)
IDH1/2	18 (25)
NPM1	9 (13)

* Cytogenetics risk groups defined in 2014 NCCN guidelines, v 2.0

† Mutation data missing for 11 patients; percentages calculated based on the number of patients with data (n=71)

Median treatment duration: 4.2 months

Median number of therapy cycles: 5

Treatment-emergent Adverse Events (AE)

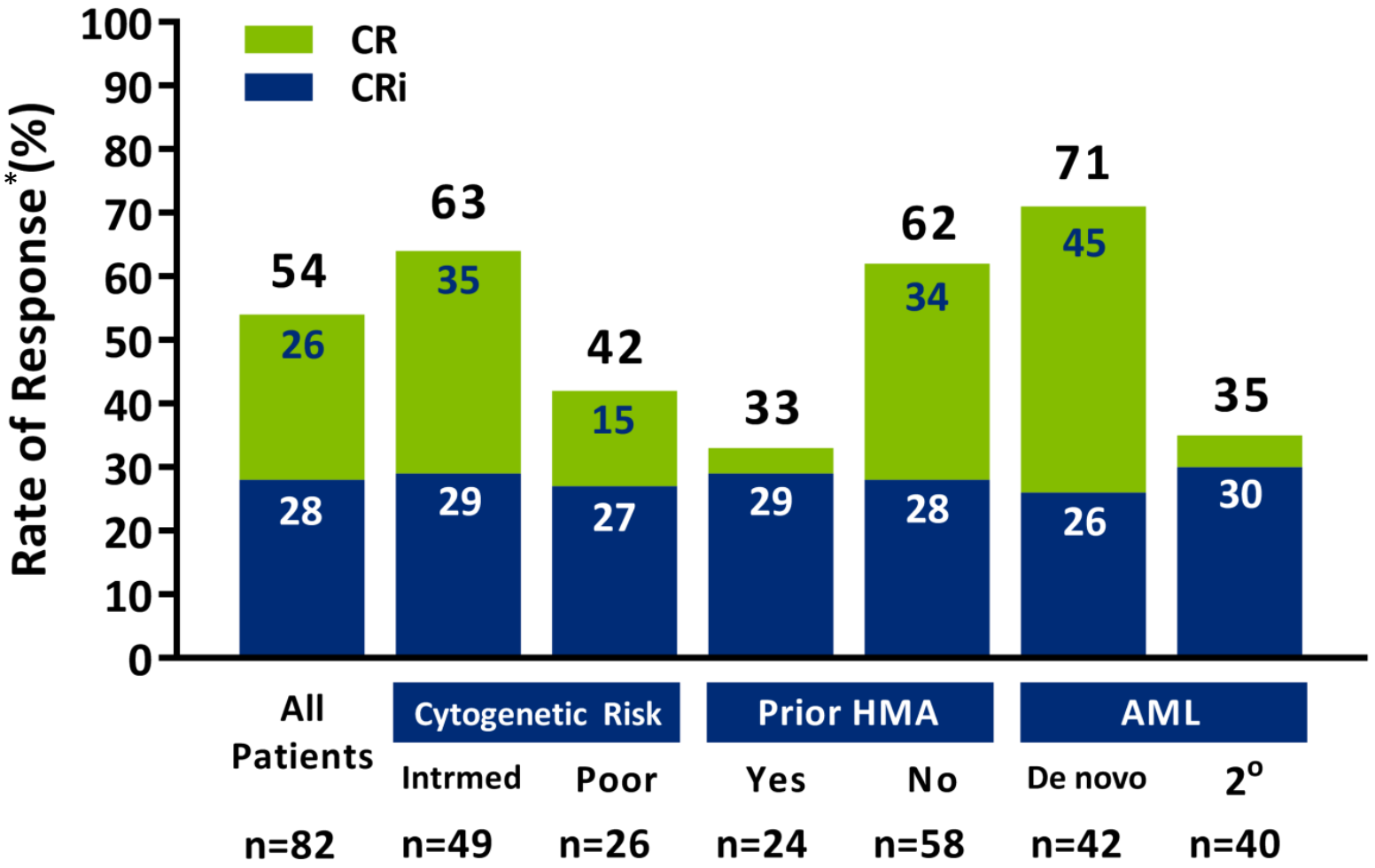
AEs in ≥30% of patients*	Any grade	Grade 3/4
Any event, n (%)	**82 (100)**	**79 (96)**
Nausea	57 (70)	2 (2)
Diarrhea	40 (49)	2 (2)
Hypokalemia	39 (48)	12 (15)
Fatigue	35 (43)	6 (7)
Febrile neutropenia	35 (43)	34 (42)
Thrombocytopenia	31 (38)	31 (38)
Constipation	29 (35)	0
Decreased appetite	28 (34)	5 (6)
WBC count decreased	28 (34)	28 (34)
Hypomagnesemia	27 (33)	1 (1)
Vomiting	25 (31)	3 (4)
Hypophosphatemia	24 (29)	13 (16)
Neutropenia	22 (27)	22 (27)
Anemia	22 (27)	22 (27)

* AEs were also listed if they were Grade ≥3 and occurred in ≥10% of patients

Serious AEs in ≥5% of patients	
Anemia	25 (31)
Febrile neutropenia	22 (27)
Pneumonia	8 (10)
Sepsis	6 (7)

Response Rates by Key Patient Subgroups

Percentage of patients with CR/CRi shown at the top of each bar



For patients with CR/CRi

Median time to first response

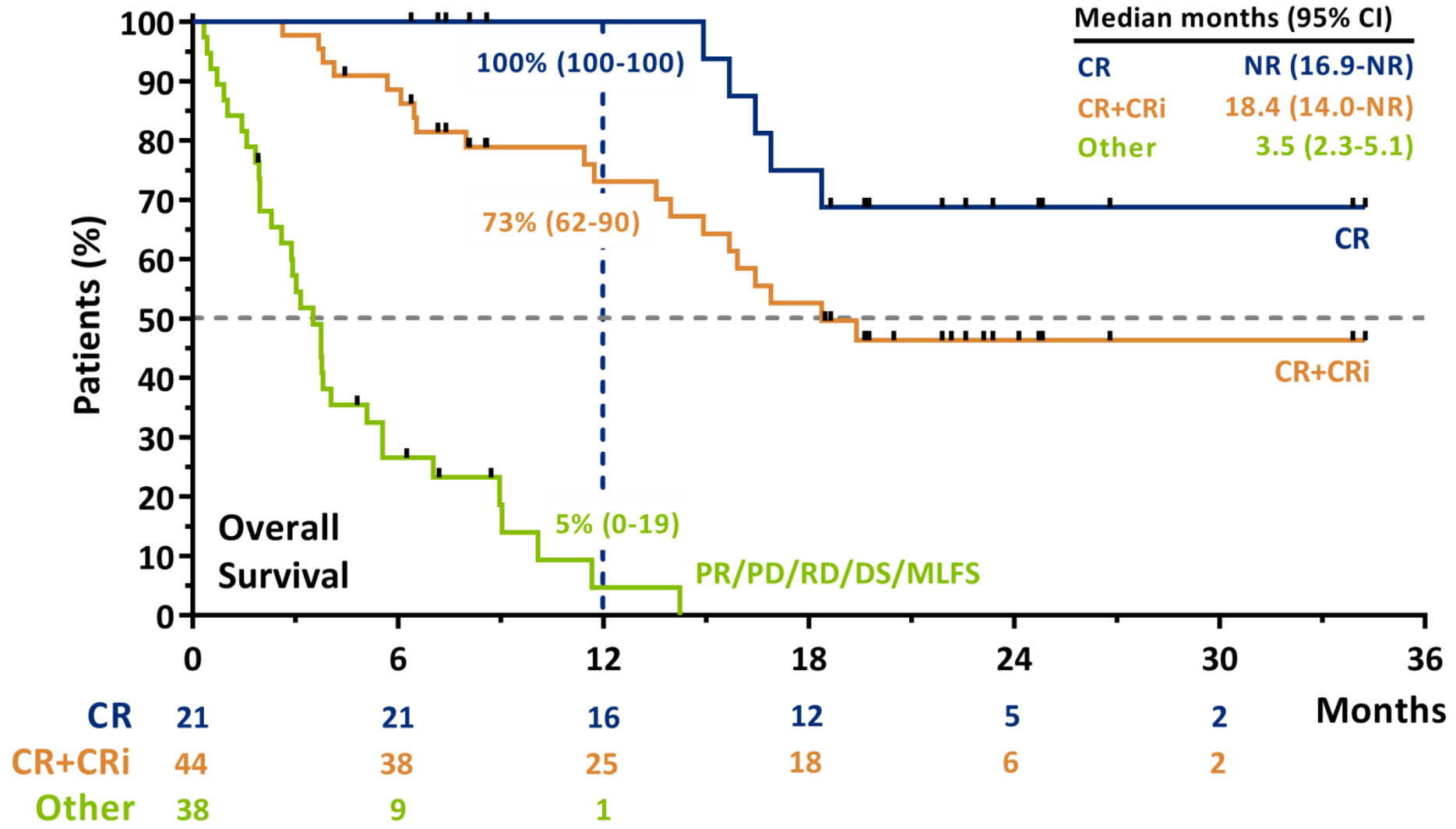
1.4 months (range 0.8–14.9)

Median time to best response

2.8 months (range 0.8–22.4)

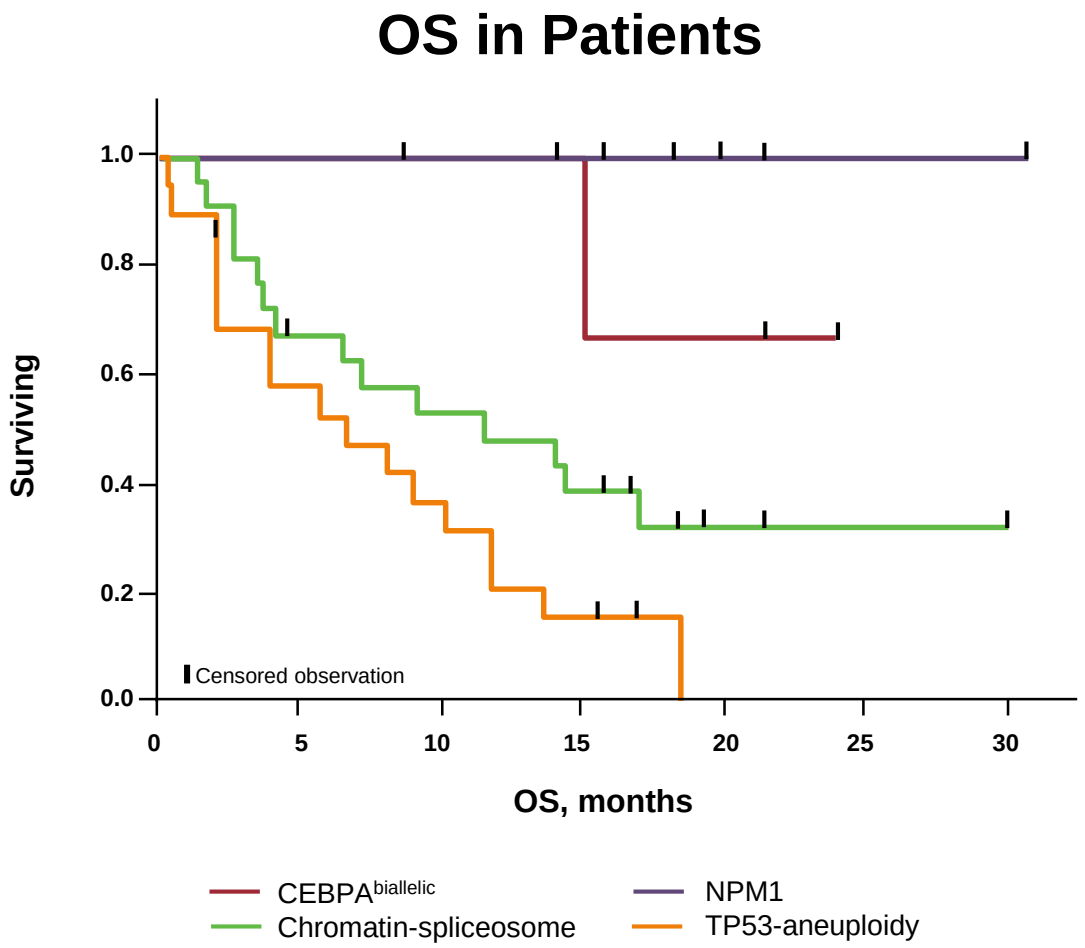
* 1 patient had a PR and 6 other patients had MLFS as best response

Overall Survival by Response



Outcomes According to Molecular Drivers of AML

Cytogenetics	ORR (CR + CRi)	Median OS, mo
Intermediate risk n = 37	28 (76%)	15.7
Adverse risk n = 19	9 (47%)	5.7
NPM1 n = 7*	7 (100%)	NR
CEBPA^{biallelic} n = 3	3 (100%)	NR
Chromatin-spliceosome n = 22	15 (68%)	11.4
TP53-aneuploidy n = 20	10 (50%)	6.5

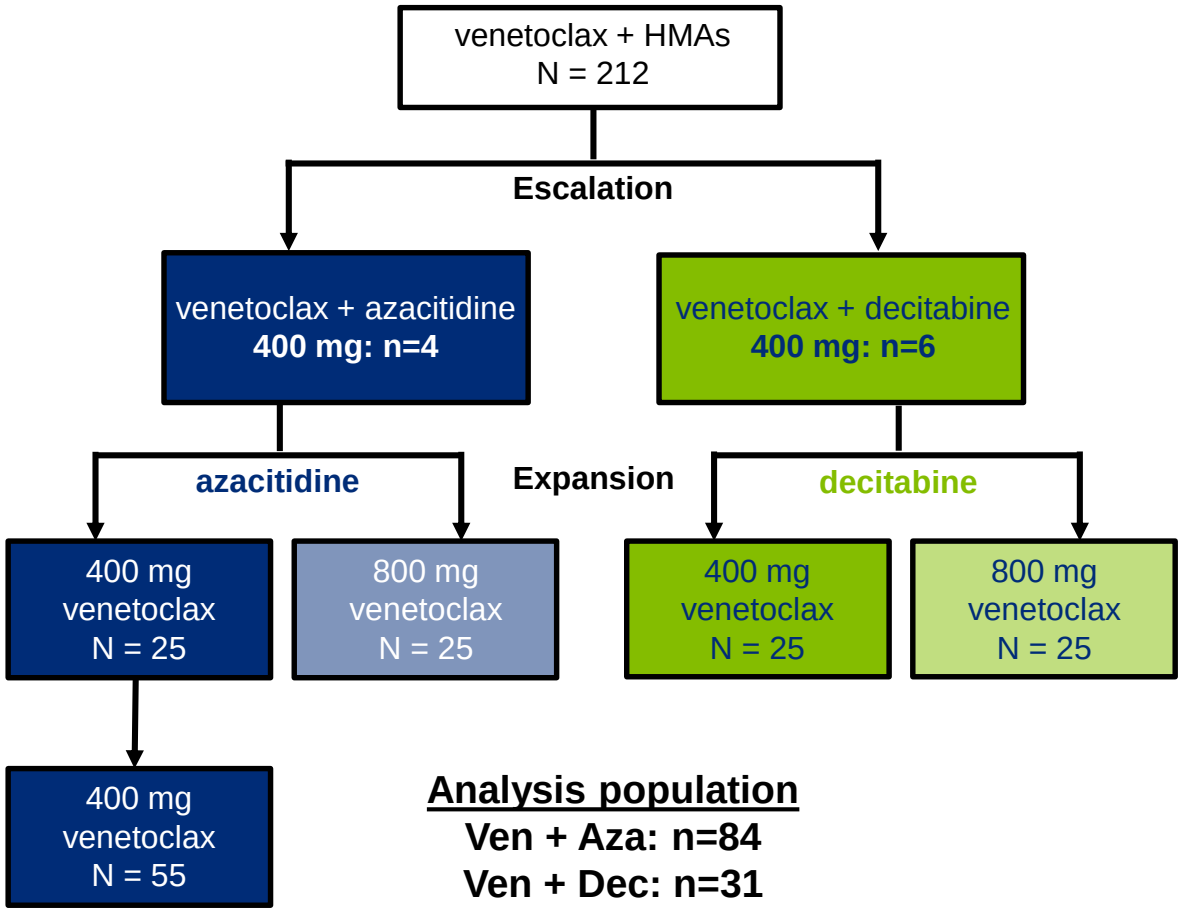


*Four of the 7 NPM1 patients have *FLT3* mutations (3: ITD, 1: TKD).

AML, acute myeloid leukemia; CR, complete remission; CRi, CR with incomplete blood count recovery; DOR, duration of response; DS, discontinued prior to assessment; NR, not reached; OS, overall survival; PD, progressive disease; RD, resistant disease.

Data cutoff date: 15 AUG 2017.

Study Overview (HMA combined with Venetoclax)



Analysis population
Ven + Aza: n=84
Ven + Dec: n=31

blood®
Leading the way in experimental and clinical research in hematology

Venetoclax combined with decitabine or azacitidine in treatment-naive, elderly patients with acute myeloid leukemia

Courtney D. DiNardo, Keith Pratz, Vinod Pullarkat, Brian A. Jonas, Martha Arellano, Pamela S. Becker, Olga Frankfurt, Marina Konopleva, Andrew H. Wei, Hagop M. Kantarjian, Tu Xu, Wan-Jen Hong, Brenda Chyla, Jalaja Potluri, Daniel A. Pollyea, and Anthony Letai

Blood 2018 :blood-2018-08-868752; doi: https://doi.org/10.1182/blood-2018-08-868752

CR/CRi rate of 67% in older AML patients with venetoclax + HMAs

Median DOR was 11.3 months and median OS was 17.5 months

400 mg venetoclax was the recommended phase 2 dose

Patient Characteristics

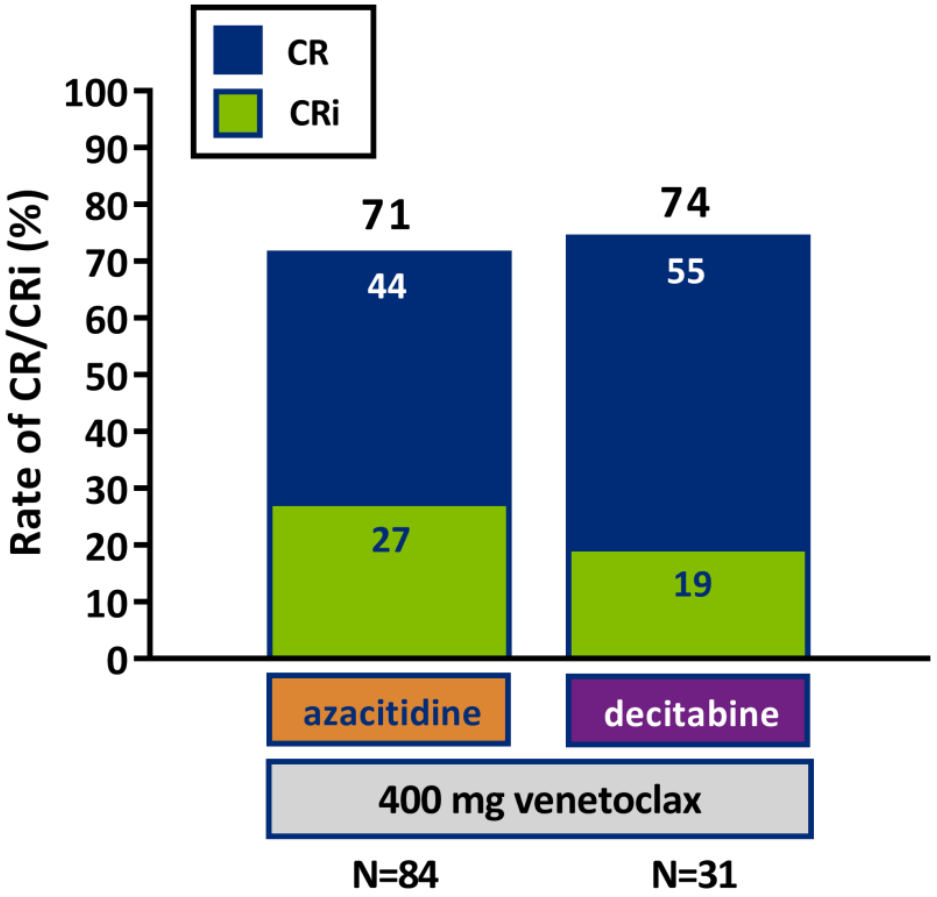
Characteristic	Venetoclax 400 mg + Aza n = 84	Venetoclax 400 mg + Dec n = 31
Median age (range), years	75 (61–90)	72 (65–86)
≥75 years, n (%)	42 (50)	8 (26)
ECOG Performance Score*, n (%)		
0–1	58 (69)	27 (87)
2	24 (29)	4 (13)
Baseline bone marrow blasts, n (%)		
<30%	24 (29)	7 (23)
≥31 – <50%	29 (34)	14 (45)
≥50%	31 (37)	10 (30)
Mutational Analyses, mutated/tested (%)		
TP53	20/74 (27)	7/22 (32)
IDH1/2	20/74 (27)	5/22 (23)
FLT3	11/74 (15)	3/22 (14)
NPM1	14/74 (19)	3/22 (14)
Cytogenetic risk†, n (%)		
Intermediate	50 (60)	16 (52)
Poor	33 (39)	15 (48)
Secondary AML, n (%)	21 (25)	9 (29)

* Two patients treated with azacitidine had an ECOG performance score of 3

† As defined by the National Comprehensive Cancer Network (NCCN) risk categorization v.2014; 1 patient in Aza cohort had no mitosis; favorable risk excluded by FISH

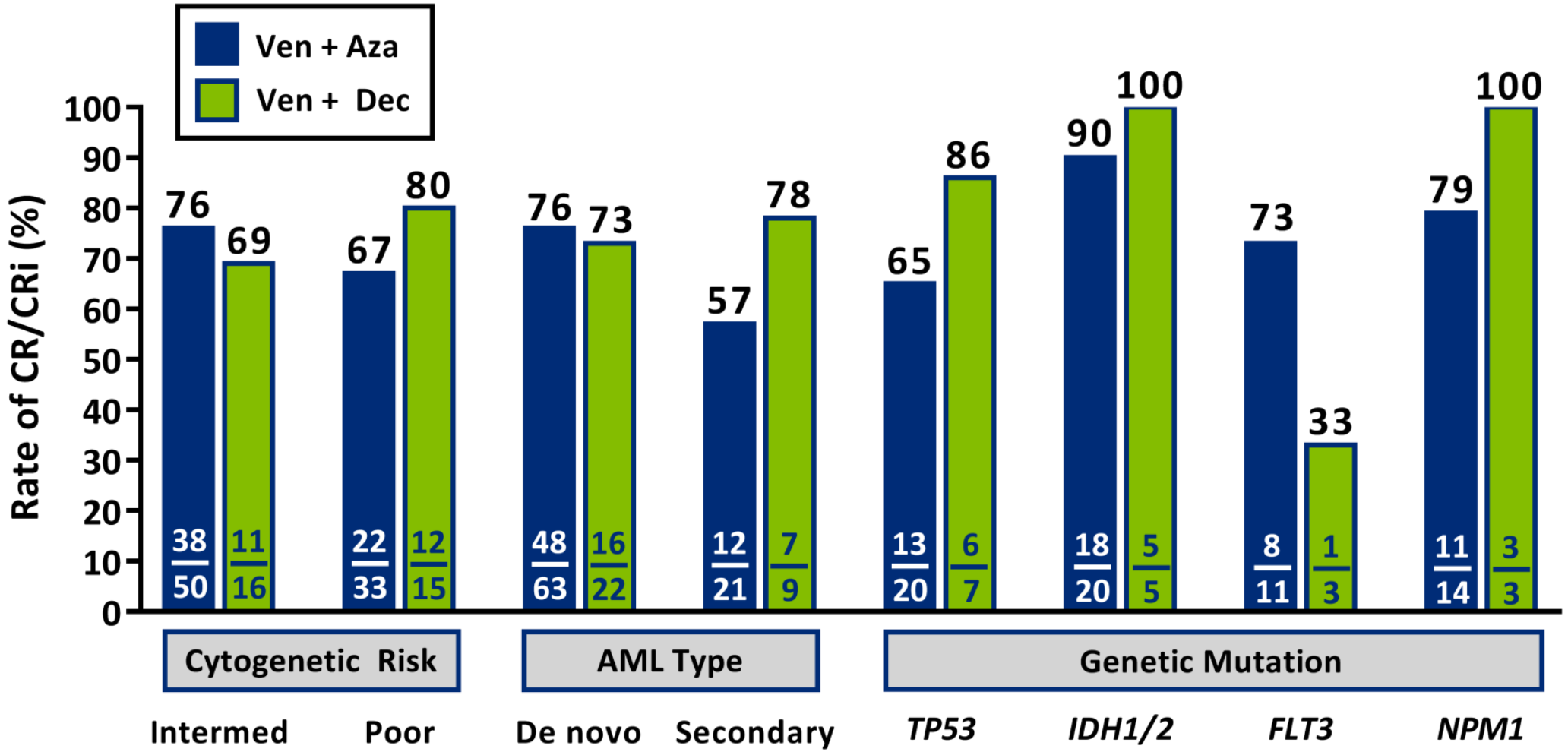
Data Cutoff Aug 31, 2018

Response Rates of CR/CRi by Combination



	Ven + Aza	Ven + Dec
Time to CR		
median (range)	1.2 (0.7–5.5)	1.9 (0.9–4.6)
No. of treatment cycles for these patients		
median (range)	6.0 (1–32)	6.0 (1–29)

Response Rates of CR/CRi by Patient Subgroups

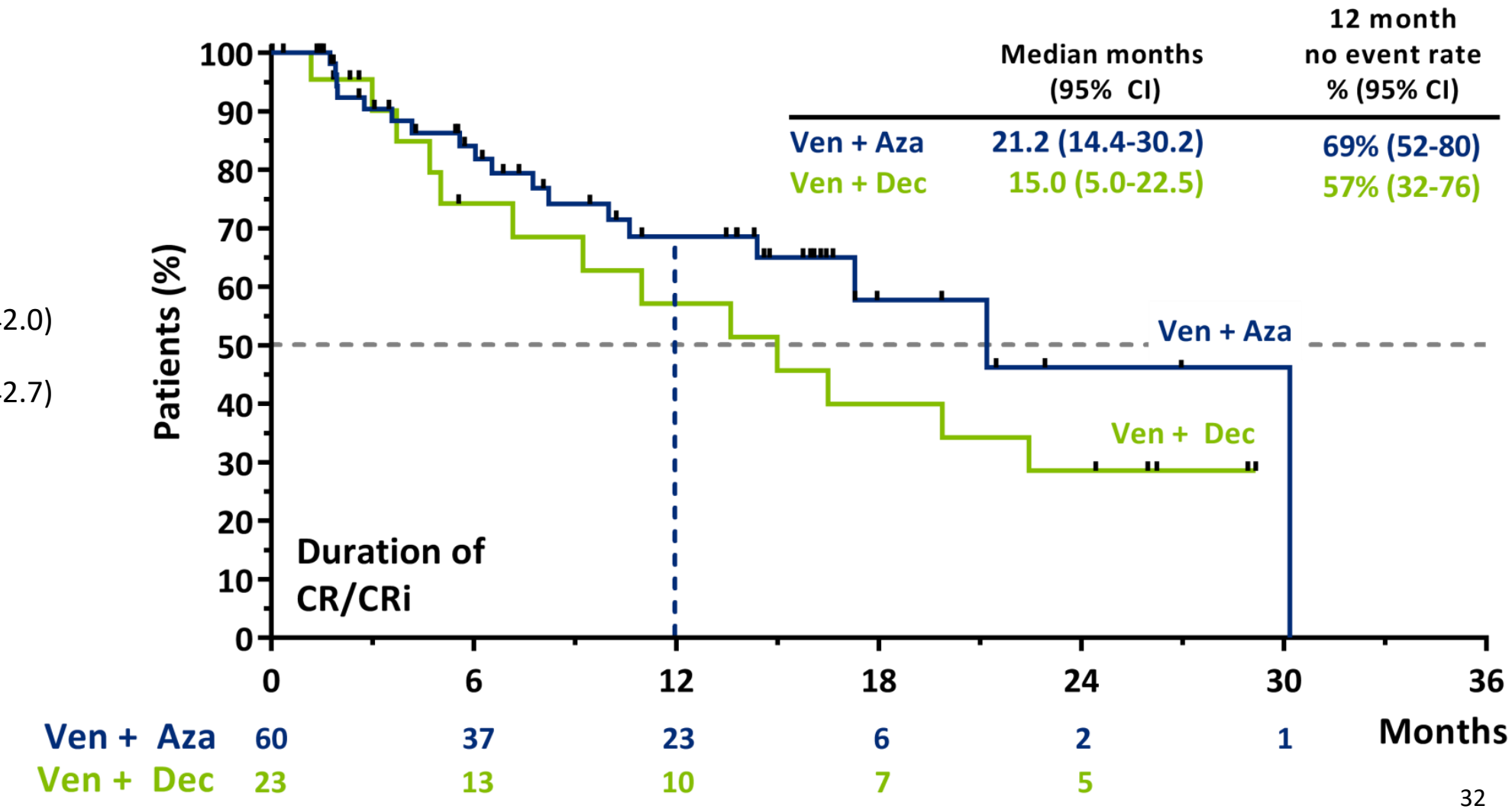


Duration of Response After Achieving CR/CRi

Median Follow-up

Venetoclax + azacitidine
14.9 months (range 0.4–42.0)

Venetoclax + decitabine
16.2 months (range 0.7–42.7)

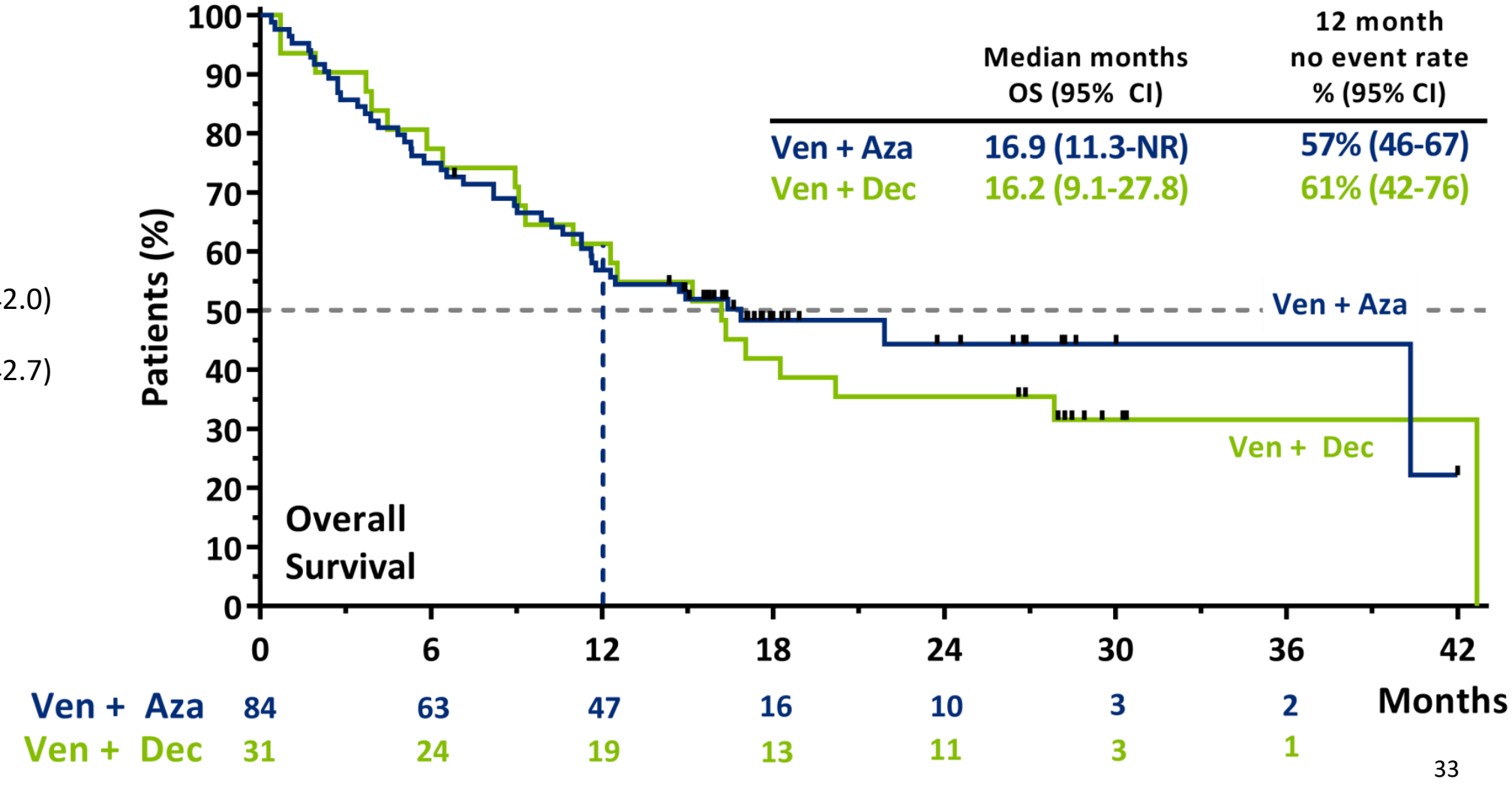


Overall Survival

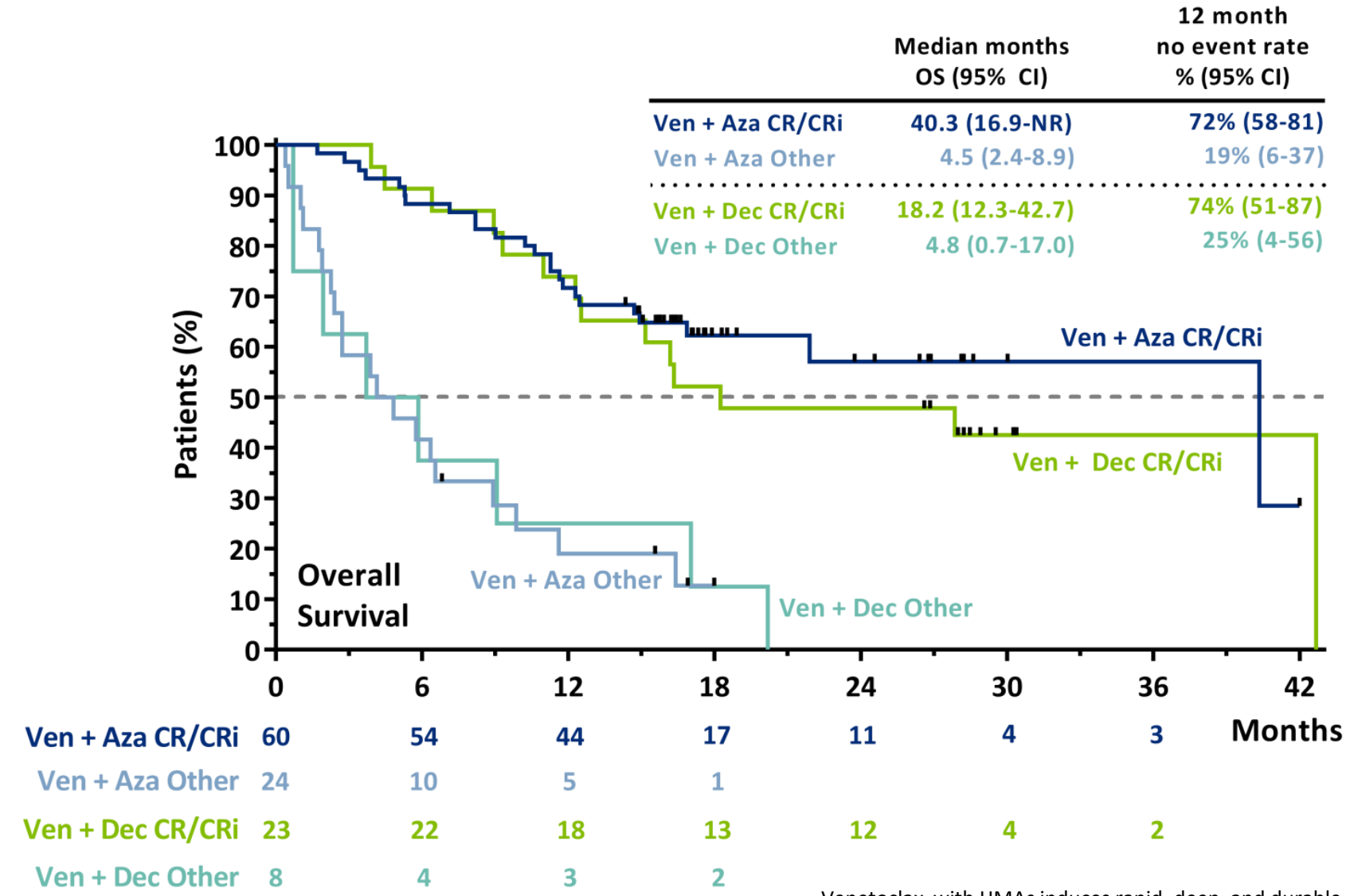
Median Follow-up

Venetoclax + azacitidine
14.9 months (range 0.4–42.0)

Venetoclax + decitabine
16.2 months (range 0.7–42.7)



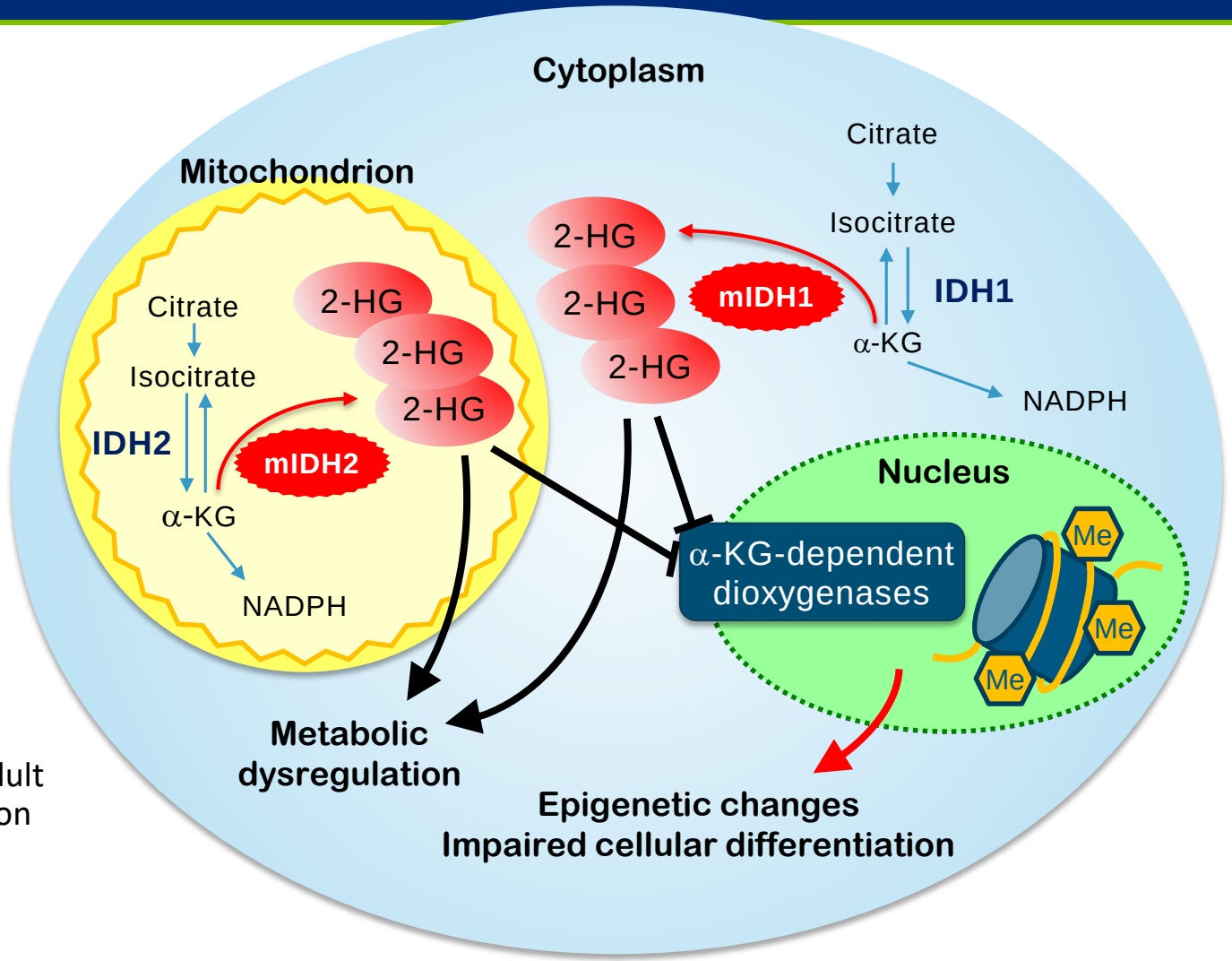
Overall Survival: CR/CRi vs. Other Responses



Other responses grouped were:
Morphological leukemia free state (MLFS), progressive disease, resistant disease

Isocitrate dehydrogenase (IDH) mutations as a target in AML

- Somatic IDH1 and IDH2 mutations result in accumulation of oncometabolite 2-HG
  - → epigenetic changes, impaired cellular differentiation
- mIDH identified in multiple solid and hematologic tumors
  - mIDH1 in ~6–10% of patients with AML
- **Ivosidenib (AG-120):** a first-in-class, oral, potent, reversible, targeted inhibitor of the mIDH1 enzyme
  - FDA approved on July 20, 2018 for the treatment of adult patients with R/R AML with a susceptible IDH1 mutation as detected by an FDA-approved test
  - under evaluation in multiple clinical trials as a single agent and in combinations



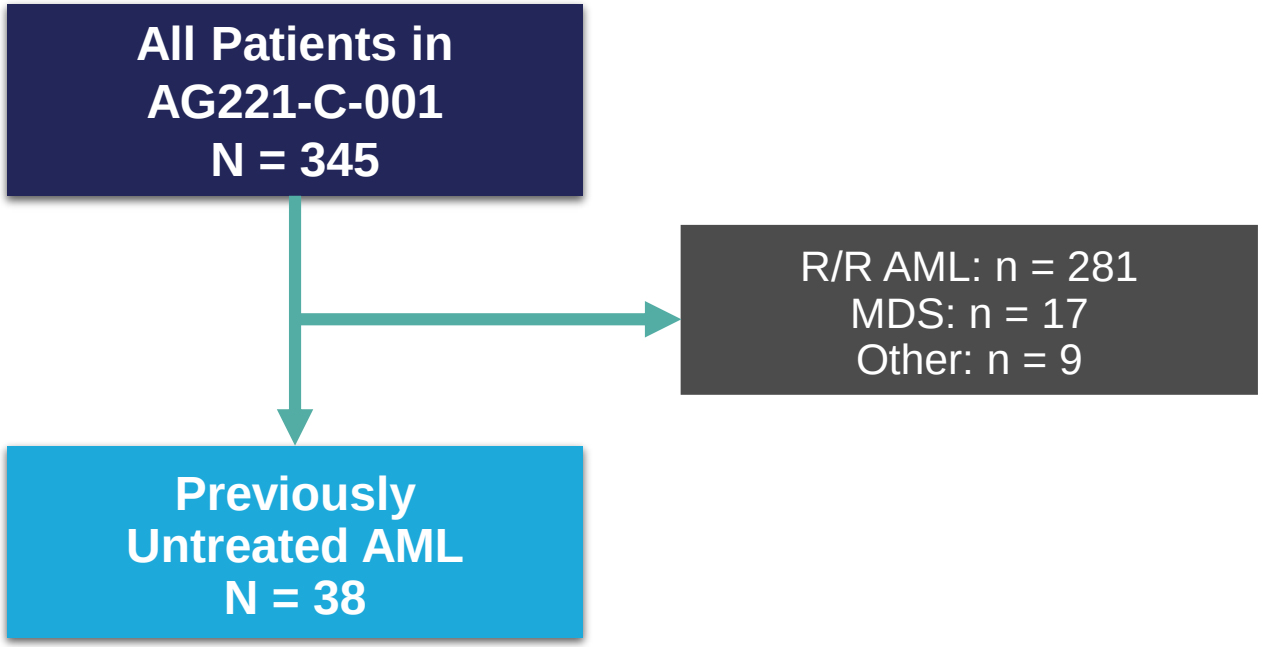
2-HG = 2-hydroxyglutarate; mIDH = mutant IDH; R/R = relapsed/refractory

OLDER PATIENTS WITH PREVIOUSLY UNTREATED *IDH2*-POSITIVE AML WERE ELIGIBLE TO ENROLL IN PHASE 1 OF THE PIVOTAL STUDY

- A subgroup of older patients with previously untreated m*IDH2* AML received enasidenib monotherapy in the phase 1 portions of the AG221-C-001 study*

- Patients:
  - Untreated m*IDH2* AML
  - ECOG PS 0-2
  - Not candidates for standard treatment

- Enasidenib dosing:
  - Dose-escalation: 50-650 mg/day
  - Expansion phase: 100 mg QD
  - Continuous 28-day treatment cycles



*NCT01915498
Data cutoff: 1 Sept 2017
ECOG PS, Eastern Cooperative Oncology Group performance status

TREATMENT-RELATED TREATMENT-EMERGENT ADVERSE EVENTS (TEAEs)

- 2 patients discontinued because of a treatment-related TEAE (cardiac tamponade, thrombocytopenia)
- Serious treatment-related TEAEs in >1 patient were IDH differentiation syndrome (n=4) and tumor lysis syndrome (n=2)

Treatment-related TEAEs	Previously Untreated m*IDH2* AML N=38		All study patients N=239[1]
	Any grade (≥10% of pts)	Grade 3-4	Grade 3-4
	n (%)		n (%)
Hyperbilirubinemia	12 (32)	5 (13)	29 (12)
Nausea	9 (24)	0	5 (2)
Thrombocytopenia	7 (18)	6 (16)	15 (6)
Fatigue	7 (18)	1 (3)	6 (3)
Decreased appetite	7 (18)	1 (3)	NR
Rash	7 (18)	0	NR
Anemia	6 (16)	5 (13)	12 (5)
IDH differentiation syndrome	4 (11)	4 (11)	15 (6)
Tumor lysis syndrome	4 (11)	3 (8)	8 (3)
ECG QT prolonged	4 (11)	1 (3)	NR
Dysgeusia	4 (11)	0	NR
Peripheral neuropathy	4 (11)	0	NR
Vomiting	4 (11)	0	NR

Data cutoff: 1 Sept 2017

ECG, electrocardiogram; IDH-DS, IDH-inhibitor-associated differentiation syndrome; NR, not reported; pts, patients; R/R AML, relapsed/refractory AML; TEAE, treatment-emergent adverse event

RESPONSE

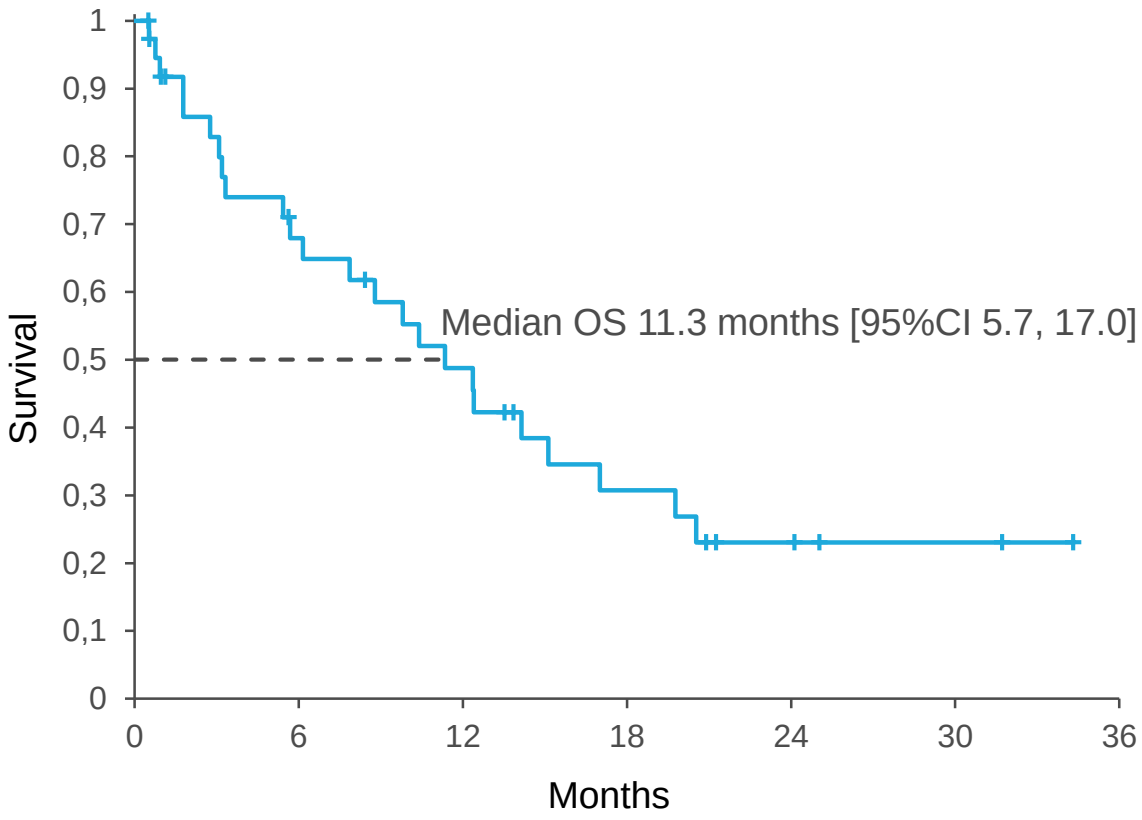
- Median number of enasidenib treatment cycles: 6.5 (range 1-35)

	Previously Untreated m*IDH2* AML N=38
Overall response (CR, CRi/CRp, PR, MLFS), n (%)	**12 (32)**
ORR 95%CI	17.5%, 48.7%
Best response, n (%)	
CR	7 (18)
CRi/CRp	1 (3)
PR	2 (5)
MLFS	2 (5)
Stable Disease*, n (%)	18 (47)
Disease Progression, n (%)	1 (3)
Not evaluable, n (%)	7 (18)

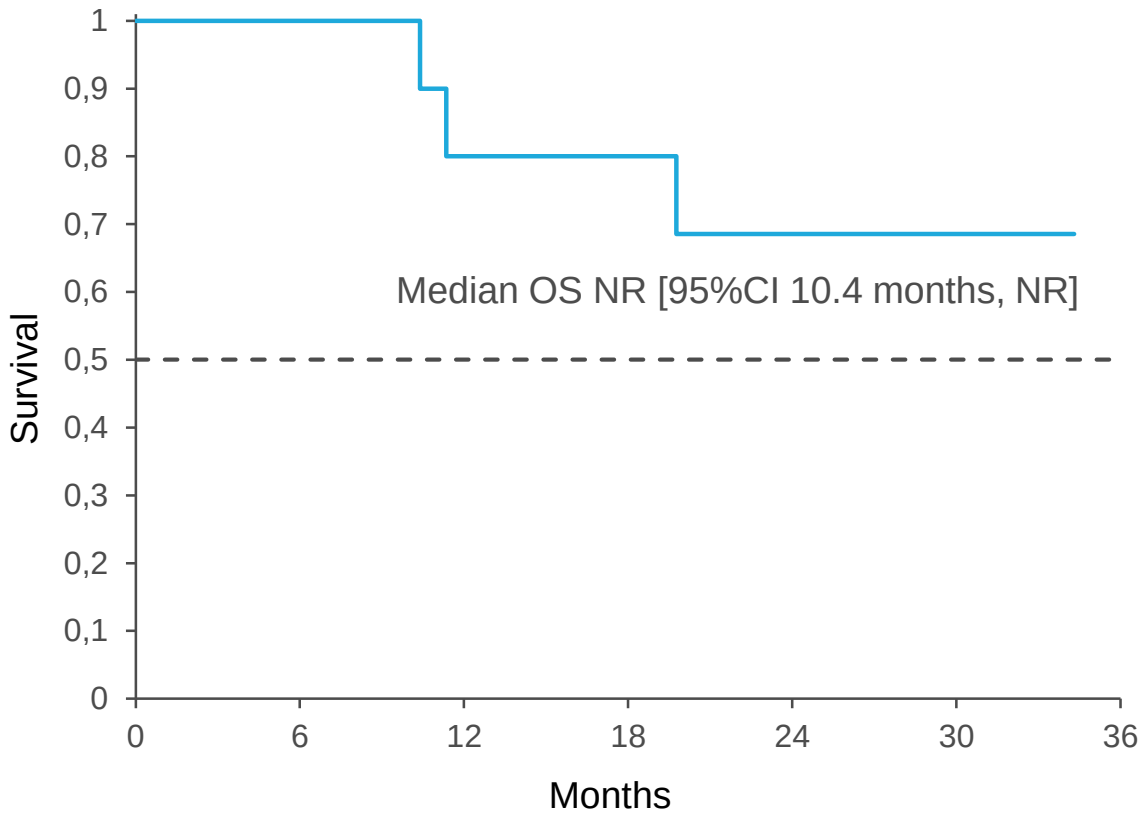
*Failure to achieve a response but not meeting criteria for progressive disease for a period of ≥8 weeks
Data cutoff: 1 Sept 2017
CR, complete remission; CRi/CRp, CR with incomplete neutrophil or platelet recovery; MLFS, morphologic leukemia-free state; ORR, Overall response rate; PR, partial remission;

OVERALL SURVIVAL

Overall Survival



Overall Survival: Responders



Data cutoff: 1 Sept 2017
NR, not reached; OS, overall survival

Leukemia
https://doi.org/10.1038/s41375-018-0312-9

ARTICLE

Acute myeloid leukemia

Randomized comparison of low dose cytarabine with or without glasdegib in patients with newly diagnosed acute myeloid leukemia or high-risk myelodysplastic syndrome

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Received: 11 October 2018 / Accepted: 2 November 2018

Cytarabine ± Glasdegib in AML and MDS
Phase 2 Study Design

N=132 Stratification by good/intermediate vs poor cytogenetic risk

Previously untreated AML or high-risk MDS not fit for intensive chemotherapy

One or more of the following:[1]

- Age ≥75 years
- ECOG PS 2
- Serum creatinine >1.3 mg/dL
- Severe cardiac disease (LVEF <45%)

Randomized 2 : 1

Low-dose Cytarabine + Glasdegib

Low-dose Cytarabine

LDAC 20 mg SC twice daily, days 1-10 of each 28-day cycle

Glasdegib 100 mg orally, once daily continuously from C1D1

Primary endpoint

OS

Secondary endpoints

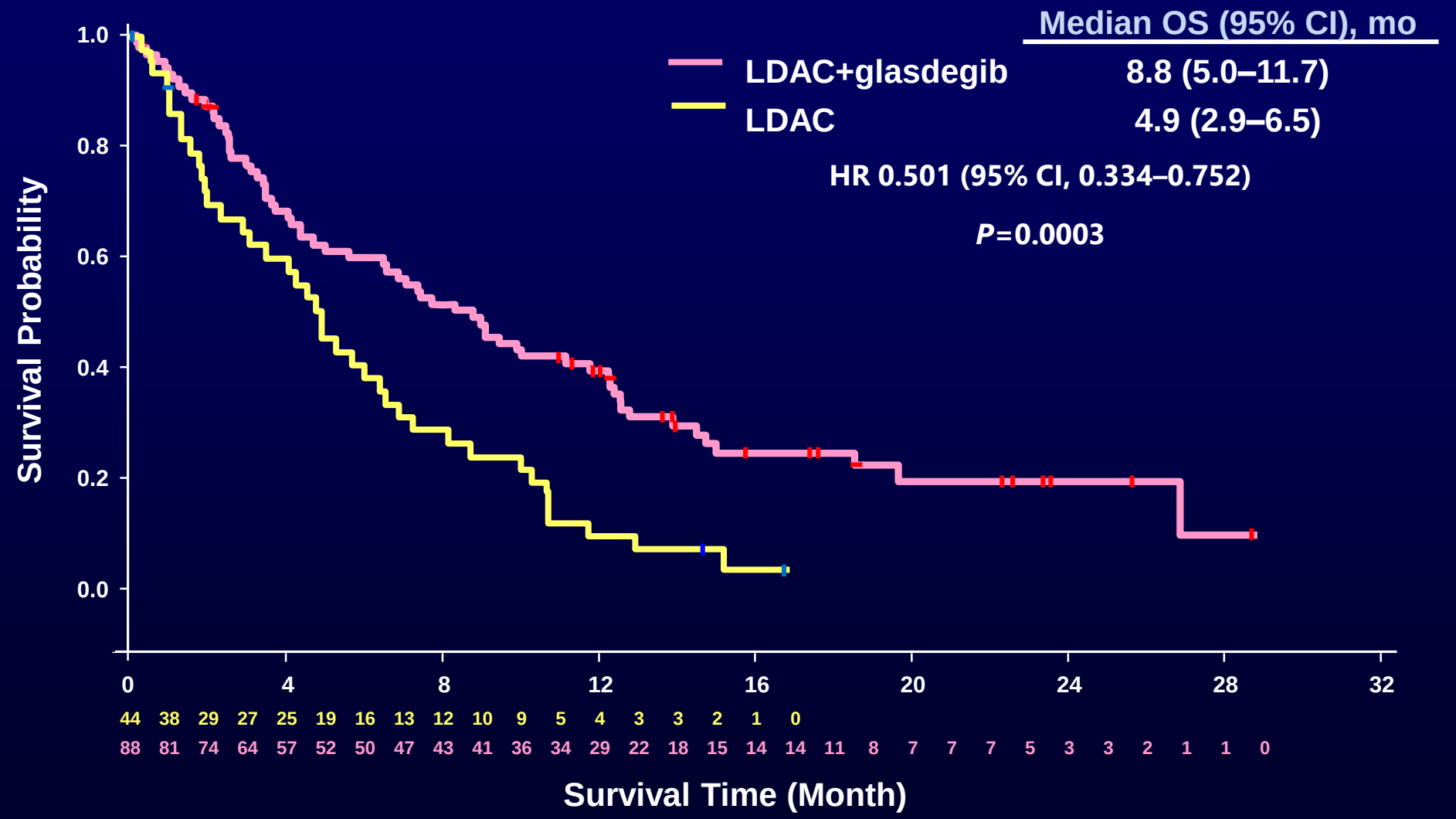
- Response
- Safety
- Pharmacodynamics
- Pharmacokinetics

[1]Kantarjian H et al. *Cancer* 2006;106(5):1090-8.

Cytarabine ± Glasdegib in AML and MDS
Patient Characteristics and Treatment Duration

Characteristic	LDAC + Glasdegib	LDAC
Randomized, N	88	44
Treated, n	84	41
Age, median (range) yrs	77 (63–92)	75 (58–83)
Diagnosis, n (%)		
AML	78 (89)	38 (86)
MDS	10 (11)	6 (14)
Cytogenetic risk, n (%)		
Good / Intermediate	55 (63)	27 (61)
Poor	33 (38)	17 (39)
Treatment duration median (range), days	83 (3–870)	47 (6–239)

Cytarabine ± Glasdegib in AML and MDS
Overall Survival



Danke für die Aufmerksamkeit
Fragen?