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Impact of demographic factors on clinical outcomes of patients with acute myeloid leukemia

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Author Conflicts of Interest

CD notes advisory board roles with EMD Serono and Cogent Biosciences. No other authors have disclosures.

Author Contributions

CD and MA designed the study question. CD submitted grant proposal, authored manuscript, and guided data analysis plan. MA assisted with data analysis plan, manuscript editing. DA conducted data analysis and authored statistical methods section. All other others assisted with manuscript review.

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Acute myeloid leukemia (AML) is an aggressive cancer with poor outcomes. Prior studies suggest non-white patients have inferior survival. This could be due to differences such as mutations in genes not previously associated with AML, higher rates of inflammation, and differences in transcriptomics.[1] Multiple studies shown Hispanic ethnicity is associated with decreased overall survival (OS).[2,3] Female sex has improved OS.[4] Whether this is applicable to community practice and the extent to which differences are due to biological or environmental factors is unclear. We utilized the Flatiron Health database and analyzed AML outcomes by factors such as race, ethnicity, sex, and socioeconomic status (SES). Given most AML patients are treated in the community, our goal was to re-examine AML outcomes by demographic factors using a real-world dataset of academic and community centers. Our goal was to expand upon previously investigated differences in clinical outcomes for AML patients given that half of AML patients are cared for in non-academic centers.[5,6]

Institutional review board approval was obtained through The University of Colorado. Data were obtained from Flatiron Health. The nationwide Flatiron Health electronic health record (EHR)-derived database is a longitudinal database, comprising de-identified patient-level structured and unstructured data, curated via technology-enabled abstraction.[7,8] The de-identified data originated from approximately 280 US-based cancer clinics (800 sites of care). Most patients in the database originate from community oncology settings. The data are deidentified and subject to obligations to prevent reidentification and protect patient confidentiality. Flatiron Health's proprietary variable for SES is created using data from the American Community Survey (2015-2019) in conjunction with the Yost Index. The SES variable is then divided into quintiles for the population based on residential address.[9] The lowest SES quintile is denoted with a 1 and the highest SES quintile is denoted with a 5.

All patients meeting study criteria were included. Inclusion criteria required patients to be age 18 or greater and carry a diagnosis of AML diagnosed via bone marrow biopsy. Patients with a diagnosis other than AML, a diagnosis of treatment related AML, or a diagnosis of acute promyelocytic leukemia were excluded. Treatment related AML was excluded given that it portends a higher risk disease state, and we were interested in examining outcomes for *de novo* AML. A minimum of two months of follow up data were required. A data cuff off date of June 30, 2022 was used for analysis.

The variable AML risk status is abstracted from chart review based on treating physician documentation. Race is a variable provided with options of White, Black, Asian, or other. Ethnicity is a variable provided with options of Hispanic or non-Hispanic. Sex is biologic sex with options for male or female. Treatment categorization is provided in Supplemental Table 1.

The primary objective was OS stratified by demographic characteristics. OS was examined by first line treatment. OS was defined as time between date of diagnosis of AML and death. Date of last visit was used for censored patients. Cox regression survival analysis was performed for univariate and multivariate analysis. Secondary

objectives included examining the effect of novel AML treatments on outcomes and assessing rate of allogenic stem cell transplantation.

3,333 patients were included (Table 1). The population was predominantly over 60 years old (70.4%), male sex (58.2%), non-Hispanic ethnicity (93.4%), and White race (75.2%). Black patients were more likely to have low SES. Asian patients had lower rates of high risk disease. White patients had higher rates of *NPM1* mutations.

With a median follow up of 3.34 years, the median OS (mOS) for the population was 15.8 months (95% CI 15, 16.9). Older age was associated with decreased survival (Figure 1). OS was assessed by race, ethnicity, sex, and SES quintile for the population (Supplemental Table 2). Patient race was not associated with a statistically significant difference in mOS. Female sex was associated with improved OS, which is consistent with prior studies.

Hispanic ethnicity was associated with improved OS compared to non-Hispanic ethnicity (22.7 months versus 15.2 months). When examining OS by race and ethnicity, Hispanic White patients had improved OS at 28.5 months (95% CI 16.7, 98) versus non-Hispanic White patients OS at 15.4 months (95% CI 14.3, 16.7) ($p = 0.006$). Samples sizes were not sufficient to analyze by Black race and ethnicity.

Lower SES was associated with inferior overall survival. mOS by SES showed improved OS for patients with high SES status (quintile 4 or 5) at 17.2 months (95% CI 15.7, 18.6) compared to patients with low SES (quintile 1 or 2) at 16.1 months (95% CI 14.1, 18.0) ($p = 0.03$).

First line treatment analysis showed greater use of 7+3 in Black versus White patients (44.7% versus 42.7%) (Table 1). This could be confounded by the younger median age of the Black patient population versus white population (58.9 years versus 65.7 years).

First line treatment by SES quintile was significant for low SES having higher rates of 7+3 (SES 1 9.7%, SES 2 8.8%) versus high SES (SES 4 5.4%, SES 5 5.2%) ($p = 0.0010$). The median age for SES 1 was 66 years and SES 2 was 67 years versus SES 4 and 5 with median age of 69 years. High SES had higher clinical trial enrollment versus low SES (SES 1 3.6%, SES 2 6.3% versus SES 4 8.3% SES 5 9.9%, $p = 0.0003$).

Given a difference in outcomes by SES, we questioned whether outcomes were impacted by access to novel therapies introduced in 2017. We hypothesized that low SES would result in decreased access to novel therapies given high out of pocket cost.

mOS for all patients was 16.5 months pre-2017 versus 15.7 months post-2017 (Figure 2A). When pre-2017 patients were assessed by SES, mOS was similar. For low SES, mOS was 17.2 months (95% CI 13.7, 21.5) and high SES mOS was 18.2 months (95% CI 14.7, 22.9) ($p=0.3534$) (Figure 2B). After 2017, low SES was associated with

decreased OS. mOS for low SES post-2017 was 15.2 months (95% CI 13.2, 17.7) versus high SES at 17 months (95% CI 15.3, 18.4) (p = 00390) (Figure 2C).

To determine what impacted OS for low SES before and after 2017 we examined the use of intensive induction therapy, novel AML agents, clinical trial enrollment and allogeneic bone marrow transplant. Pre-2017 low SES patients were less likely to receive 7+3 chemotherapy (31.2% versus 35.9%, p = 0.0052), less likely to enroll in a clinical trial (4.9% versus 11.0%, p = 0.0081), and less likely to undergo an allogeneic stem cell transplant (33.6% versus 66.4%, p = <0.0001) compared to high SES patients. Post-2017 low SES patients had higher rates of intensive induction chemotherapy, lower rates of clinical trial enrollment and had lower rates of allogeneic stem cell transplant (Supplemental Table 3). Rates of novel AML agent use were not statistically significant (24% versus 28.3%) but could be clinically significant.

This is a real world study of AML patients treated at academic and community centers. Prior studies demonstrated Black AML patients have worse OS versus White patients. Our study did not find a difference in OS by race which could have been due to small sample size. Hispanic ethnicity had improved OS, a novel finding.[10] The lower rate of CHIP among Hispanic patients may contribute to decreased incidence of MDS and subsequently, AML.[11] Female sex was associated with improved outcomes which is consistent with prior research.

The study demonstrates low SES impacts outcomes. Low SES patients had worse OS, decreased utilization of novel agents, lower rates of clinical trial enrollment and transplant which impact survival. Even in the era of post-transplant cytarabine allowing for greater donor mismatch, rates of allogenic transplant were still lower. Consistent with our findings, a study of English patients showed low SES had a negative impact on OS for AML.[12] In an analysis of Danish residents with AML, low SES status was associated with worse outcomes for AML.[13] This suggests access to insurance is not the only factor affecting outcomes. For our patient cohort, the differences in transplant and clinical trials may have impacted OS.

This analysis has its limitations. This is a retrospective review of real-world data and the analysis is impacted by patient sample sizes and data limitations. The study population was predominantly White and may not be representative of the US population. Race and SES may be confounded by other risk factors not included in the dataset. Analysis was not stratified by prior hematologic malignancy which could impact outcomes. Requiring two months of follow up could introduce bias as patients with early death may be excluded.

This analysis demonstrates the need to continue investigation on the impact of ethnicity, SES, and access to care on outcomes for AML patients.

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Tables:

Table 1. Demographic Factors of Study Population.

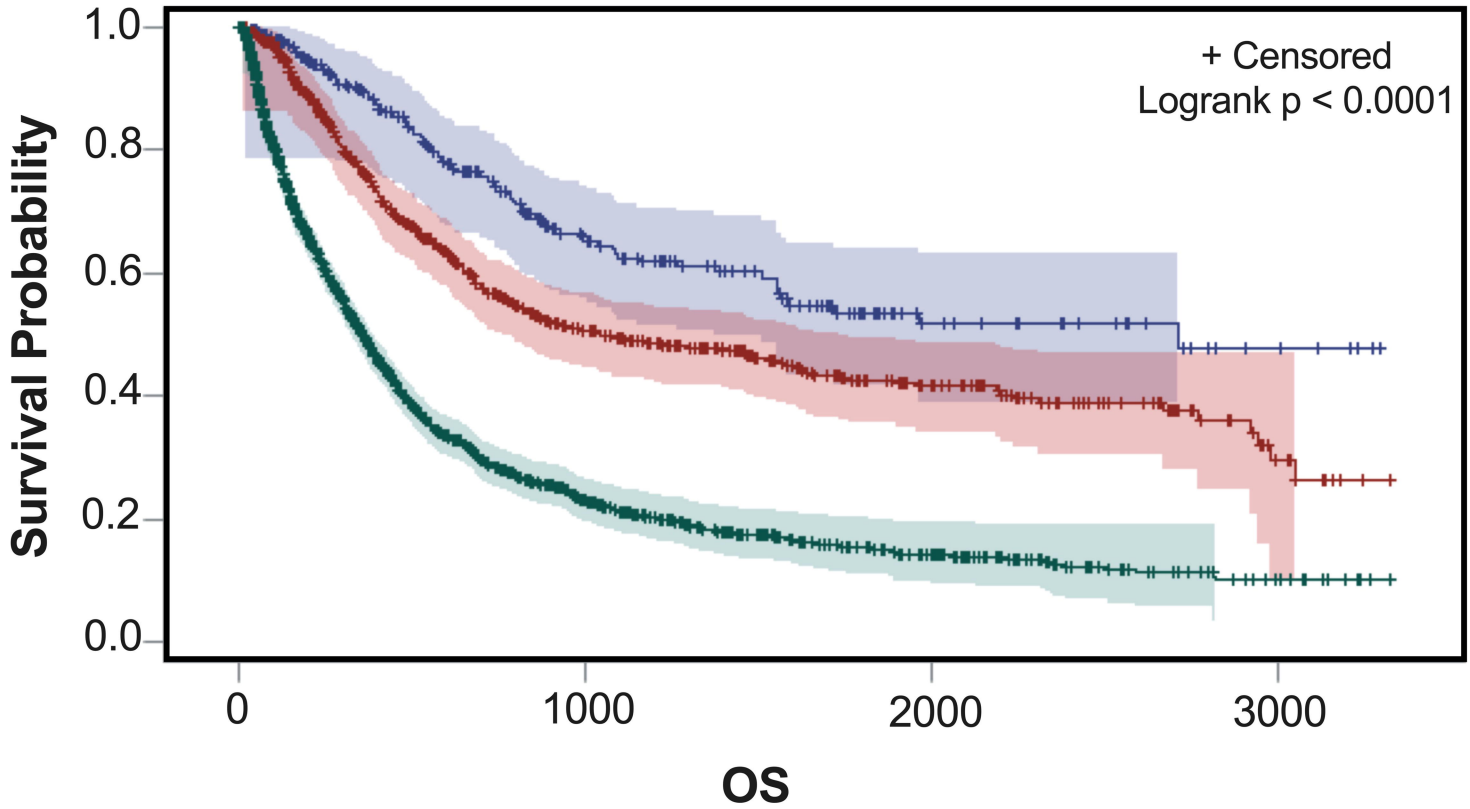
	Study population N = 3333 N (%)	Asian N = 59 (2%)	Black N = 228 (7.7%)	White N = 2241 (75.2%)	Other N = 450 (15.1%)	p-value
Age at Diagnosis: Years						
Minimum	18	24	20	18	19	
Maximum	85	85	85	85	85	
Median	68	59	61.5	69	68	
Mean	64.87	59.05	58.91	65.69	64.62	
Standard Deviation	15.02	15.04	16.19	14.51	15.47	
Age: N(%)						
18 – 39 years	279 (8.4)	6 (10.2)	36 (15.8)	163 (7.3)	39 (8.7)	<0.0001
40 – 59 years	708 (21.2)	25 (42.4)	70 (32)	444 (19.8)	100 (22.2)	
60+ years	2346 (70.4)	28 (47.5)	119 (52.2)	1634 (72.9)	311 (69.1)	
Sex: N(%)						
Female	1392 (41.8)	24 (40.7)	117 (51.3)	920 (41.1)	191 (42.4)	0.0593
Male	1941 (58.2)	35 (59.3)	111 (48.7)	1321 (58.9)	259 (57.6)	
Ethnicity: N(%)	955 (28.7)					<0.0001
Hispanic/Latino/Unknown/Missing	2378 (71.3)	11 (18.6)	56 (24.6)	357 (15.9)	230 (51.1)	
Non-Hispanic		48 (81.4)	172 (75.4)	1884 (84.1)	220 (48.9)	
SES Quintile: N(%)	999 (33.0)					<0.0001
1/2	675 (22.3)	12 (21.8)	111 (53.4)	610 (30.1)	151 (37.7)	
3	755 (24.9)	7 (12.7)	43 (20.7)	472 (23.2)	90 (22.4)	
4	598 (19.8)	12 (21.8)	36 (17.3)	525 (25.9)	98 (24.4)	
5		24 (43.6)	18 (8.7)	422 (20.8)	62 (15.5)	
ECOG N(%)	534 (28.4)					0.4010
0	921 (49.0)	11 (30.6)	40 (36.7)	368 (27.9)	73 (29.0)	
1	424 (22.6)	20 (55.6)	51 (46.8)	649 (49.2)	123 (48.8)	
2/3/4		<=5 (13.9)	18 (16.5)	302 (22.9)	56 (22.2)	
Disease Characteristics						
Karyotype: N(%)	563 (16.9)					0.0161
Favorable/Low Risk	701 (21.0)	14 (23.7)	48 (21.1)	360 (16.1)	87 (19.3)	
Intermediate Risk	2069 (62.1)	12 (20.3)	37 (16.2)	495 (22.1)	85 (18.9)	
Poor/adverse/High Risk		33 (55.9)	143 (62.7)	1386 (61.9)	278 (61.8)	
Blast Percentage at Diagnosis: N(%)	31 (0.9) 53 (1.6)					0.4311
< 1-5%	67 (2.0)	<=5 (X)	<=5 (X)	24 (1.1)	6 (1.3)	
6 - 10%	259 (7.8)	<=5 (X)	<=5 (X)	38 (1.7)	9 (2.0)	
11 - 15%	349 (10.5)	<=5 (X)	6 (2.6)	45 (2.0)	9 (2.0)	
16 - 20%	329 (9.9)	7 (11.9)	17 (7.5)	169 (7.5)	35 (7.8)	
21 - 25%	160 (4.8)	7 (11.9)	30 (13.2)	234 (10.4)	41 (9.1)	
26 - 30%	257 (7.7)	<=5 (X)	21 (9.2)	224 (10.0)	44 (9.8)	
31 - 35%	112 (3.4)	<=5 (X)	<=5 (X)	117 (5.2)	22 (4.9)	
36 - 40%	197 (5.9)	6 (10.2)	20 (8.8)	171 (7.6)	39 (8.7)	
41 - 45%	1380 (41.4)	<=5 (X)	<=5 (X)	80 (3.6)	15 (3.3)	
46 - 50%	139 (4.2)	<=5 (X)	17 (7.5)	129 (5.7)	26 (5.8)	
> 50%		22 (37.3)	94 (41.2)	918 (41)	193 (42.9)	
Unknown		6 (10.2)	12 (5.3)	92 (4.1)	11 (2.4)	
Mutation Status: N(%)						
ANKRD26 (N = 156 Tested)	7 (4.5)	<=5 (X)	<=5 (X)	<=5 (X)	<=5 (X)	**0.026**
ASXL1 (N = 1472 Tested)	317 (21.5)	<=5 (X)	20 (21.1)	214 (22.1)	51 (23.6)	0.932
CALR (N = 1068 Tested)	16 (1.5)	<=5 (X)	<=5 (X)	13 (1.9)	<=5 (X)	0.0935

CEBPA (N = 1948 Tested)	164 (8.4)	<=5 (X)	16 (12.8)	107 (8.3)	24 (8.3)	0.2942
CSF3R (N = 1263 Tested)	28 (2.2)	<=5 (X)	<=5 (X)	15 (1.8)	7 (3.5)	0.0649
DDX41 (N = 349 Tested)	17 (4.9)	<=5 (X)	<=5 (X)	17 (7.4)	<=5 (X)	0.0810
DNMT3A (N = 1578 Tested)	457 (29)	<=5 (X)	26 (26.3)	321 (30.6)	54 (24.3)	0.1285
ETV6 (N = 1277 Tested)	47 (3.7)	<=5 (X)	<=5 (X)	36 (4.3)	7 (3.7)	0.7529
EZH2 (N = 1353 Tested)	73 (5.4)	<=5 (X)	<=5 (X)	53 (5.9)	9 (4.4)	0.7878
FLT3 (N = 1927 Tested)	218 (11.3)	<=5 (X)	8 (6.3)	145 (11.4)	34 (12.2)	0.936
FLT3 ITD (N = 2145 Tested)	403 (18.8)	10 (23.8)	19 (13.2)	278 (19)	40 (14.7)	0.778
FLT3 TKD (N = 1958 Tested)	170 (8.7)	2 (5)	6 (4.5)	120 (8.9)	23 (9.2)	0.3682
GATA2 (N = 1038 Tested)	61 (5.9)	2 (16.7)	4 (6.5)	43 (6.4)	8 (5.1)	0.3850
IDH1 (N = 1872 Tested)	206 (11)	3 (9.1)	12 (10.6)	146 (11.7)	23 (8.6)	0.5441
IDH2 (N = 1897 Tested)	310 (16.3)	6 (17.7)	21 (18.3)	193 (15.3)	53 (19.8)	0.2668
JAK2 (N = 1414 Tested)	85 (6)	0 (0)	6 (7.1)	58 (6.2)	14 (6.7)	0.9044
KIT (N = 1596 Tested)	97 (6.1)	1 (3.9)	8 (7.8)	59 (5.6)	20 (8.5)	0.3343
KRAS (N = 1366 Tested)	104 (7.6)	1 (6.7)	11 (13.7)	72 (8)	12 (5.7)	0.1543
MPL (N = 1121 Tested)	13 (1.2)	0 (0)	1 (1.5)	10 (1.4)	2 (1.2)	1.000
NF1 (N = 659 Tested)	59 (8.9)	2 (25)	2 (4.3)	40 (9)	7 (8.1)	0.2577
NPM1 (N = 2245 Tested)	456 (20.3)	7 (16.3)	15 (10.9)	319 (21.1)	52 (16.5)	**0.0012**
NRAS (N = 1449 Tested)	252 (17.4)	5 (25)	22 (25.3)	164 (17.2)	34 (15.7)	0.1679
RUNX1 (N = 1547 Tested)	343 (22.2)	4 (22.2)	30 (30)	223 (21.9)	51 (21.5)	0.3161
SRP72 (N = 96 Tested)	1 (1)	0 (0)	0 (0)	0 (0)	1 (5.6)	0.2472
TET2 (N = 1487 Tested)	396 (26.6)	3 (17.7)	23 (25.3)	283 (28.6)	54 (24.7)	0.5058
TP53 (N = 1509 Tested)	410 (27.2)	3 (16.7)	22 (24.4)	264 (26.5)	73 (32.5)	0.2147
Treatment Information						
1L Treatment: N(%)						
HMA	718 (21.5)	<=9 (X)	36 (15.8)	500 (22.3)	89 (19.8)	<0.000
7+3	1415 (42.5)	34 (57.6)	102 (44.7)	958 (42.7)	216 (48.0)	1
Clinical Trial	222 (6.7)	<=9 (X)	17 (7.5)	166 (7.4)	16 (3.6)	
Novel AML Therapy	578 (17.3)	<=9 (X)	29 (12.7)	379 (16.9)	99 (22.0)	
Other	400 (12.0)	<=9 (X)	44 (19.3)	238 (10.6)	30 (6.7)	
Intensive Chemotherapy: N(%)						0.0022
Yes	894 (33.6)	25 (54.3)	60 (35.1)	566 (32.0)	119 (34.2)	
No	1542 (58.0)	19 (41.3)	94 (55.0)	1038 (58.6)	213 (61.2)	
Unknown	222 (8.3)	2 (4.3)	17 (9.9)	166 (9.4)	16 (4.6)	
Allogeneic HSCT: N(%)						0.0063
Yes	854 (25.6)	14 (23.7)	49 (21.5)	617 (27.5)	90 (20.0)	
No	2479 (74.4)	45 (76.3)	179 (78.5)	1624 (72.5)	360 (80.0)	
Novel Therapeutic Agent: N(%)						0.0013
Yes	909 (46.4)	14 (38.9)	52 (38.8)	590 (45.9)	138 (52.3)	
No	844 (43.1)	21 (58.3)	65 (48.5)	538 (41.9)	110 (41.7)	
Unknown	207 (10.6)	1 (2.8)	17 (12.7)	156 (12.1)	16 (6.1)	

Figure Legends

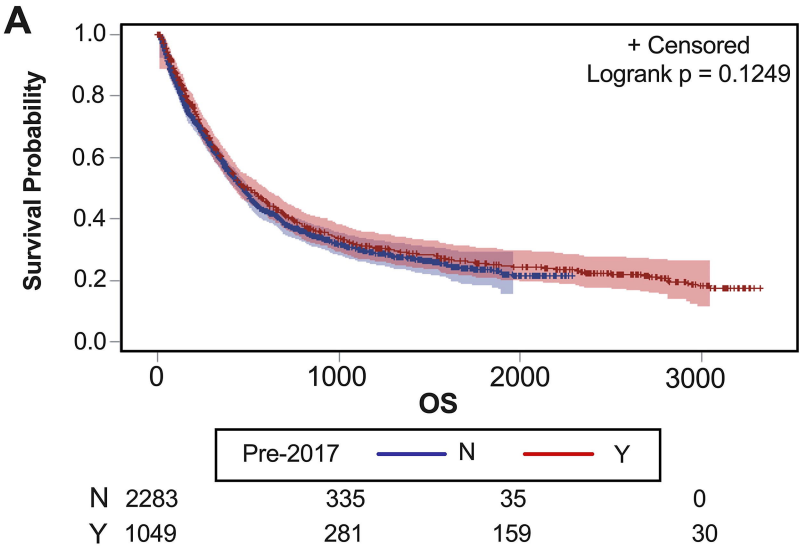
Figure 1. Median Overall Survival by Age Group. Median overall survival was assessed by prespecified age groups. Survival for patients age 18-39 years (blue) was 90.3 months (95% CI 51.9, not reached), for patients aged 40-59 years (red) was 35.3 months (95% CI, 27.4, 51.9) and for patients aged ≥ 60 (green) was 11.9 months (95% CI, 11.2, 12.6)

Figure 2. Median Overall Survival by Year of Diagnosis and SES. Panel A: Median overall survival was assessed by year of diagnosis for either before 2017 (red) or after 2017 (blue). mOS for diagnosis before 2017 was 16.5 months (95% CI 14.6, 18.9) compared to mOS for diagnosis after 2017 which was 15.7 months (95% CI 14.7, 16.8) p =0.1249. Panel B: Median overall survival by low (blue) versus high (red) SES pre-2017. Panel C: Median overall survival by low (blue) vs high (red) SES post-2017.



Age Group — 18 - 40 years — 40 - 60 years — > = 60 years

18 - 40 years	279	102	27	6
40 - 60 years	707	203	76	11
> = 60 years	2346	311	91	13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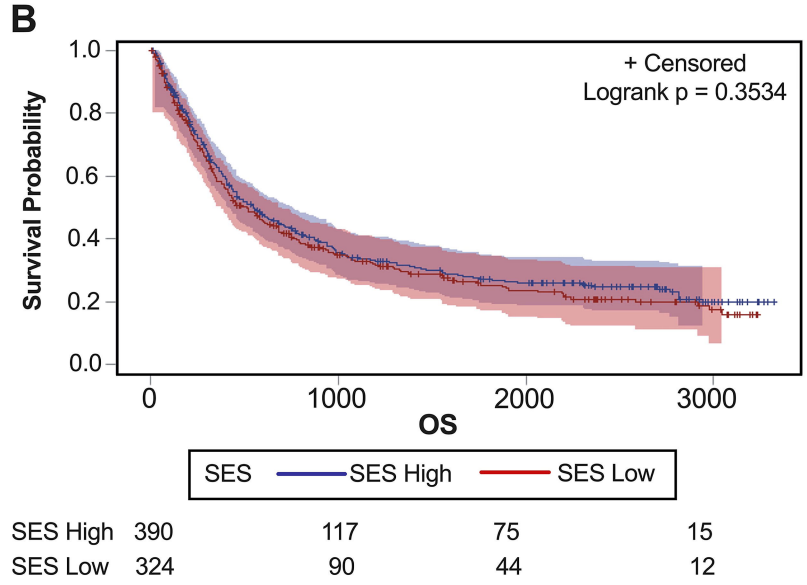
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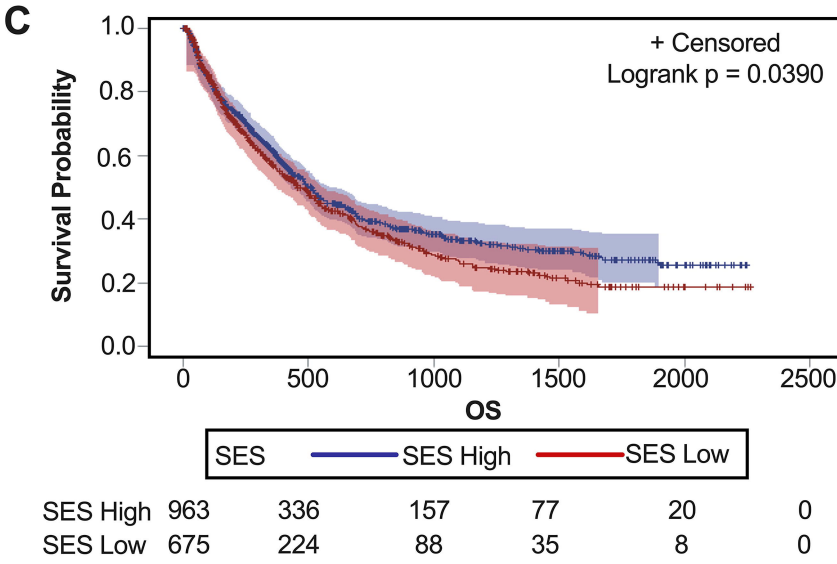
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SES Low 675 224 88 35 8 0

Supplemental Figures and Tables

Supplementary Table 1. Classification of 1L Treatments

1L Treatment Categorization	**Examples of Treatments**
HMA	Azacitidine, Decitabine
7+3	7+3, CPX351
Clinical Trial	Clinical study drug used in treatment per Flatiron Health
Novel AML Therapy	Venetoclax, gemtuzumab ozogamicin, midostaurin, gilteritinib, ivosidenib, enasidenib, glasdegib
Other	Cyclophosphamide, bosutinib, dasatinib, single agent hydroxyurea, reteinoin, sorafenib, ruxolitinib, lenalidomide

Supplementary Table 2. Median OS by Demographic Characteristics

Characteristic	Study Population Median OS (95% CI) P value	Age 18-39 Median OS (95% CI) P value	Age 40-59 Median OS (95% CI) P value	Age ≥ 60 Median OS (95% CI) P value
Race				
Asian	21.4 months (12.6, 23)	28.1 months, (2.3, NR)	22.6 months (13.7, NR)	12.2 months (6.5, 17.4)
Black	18.4 months (15, 24)	33.5 months (20.8, 57.4)	31.9 months (20.6, 101.6)	12.5 months (9.8, 14.9)
White	16.1 months (14.9, 17.1)	NR (65.3, NR)	49.6 months (28.3, 74.6)	12.3 months (11.2, 12.6)
Other	15.4 months (13.6, 18.2)	52.8 months (20.7, NR)	30 months (22.2, 52.6)	12.2 months (9.9, 14.8)
	P = 0.6686	P = 0.0612	P = 0.8165	P = 0.6649
Ethnicity				
Hispanic/Latino	22.7 months (17.4, 35.9)	NR (26.7, NR)	28.5 months (17.4, 98)	16.8 months (12.5, 20.1)
Non-Hispanic/Latino	15.2 months (14.4, 16.4)	90.3 months (45.9, NR)	34.2 months (26.3, 52.7)	11.7 months (10.8, 12.5)
	P = 0.001	P = 0.8286	P = 0.7364	P = 0.0847
Sex				
Male	15 months (13.8, 16.1)	65.3 months (38.5, NR)	31.2 months (22.7, 48.8)	11.4 months (10.4, 12.5)
Female	17.7 months (15.6, 19.3)	90.3 months (51.9, NR)	48.9 months (28.5, 73)	12.4 months (11.6, 13.5)
	P < 0.0001	P = 0.5038	P = 0.0610	P = 0.0162
SES Quintile				
Low SES (1 + 2)	16.1 months (14, 18)	52.8 months (31, NR)	35.3 months (23.2, 57.9)	10.8 months (9.4, 12)
High SES (4 + 5)	17.2 months (15.7, 18.6)	NR (52.2, NR)	51.9 months (27.7, 92.2)	13.3 months (12.1, 14.5)
	P = 0.0311	P = 0.1967	P = 0.1699	P = 0.0009

Supplementary Table 3. Rates of Post-2017 Treatments by SES.

Therapy	Low SES (1/2)	High SES (4/5)	P-value
Induction chemotherapy (7+3)	43%	35.7%	0.0013
Novel AML Agents (TKI, HMA/Venetoclax etc)	24%	28.3%	0.0553
Single Agent HMA	17.3%	17.5%	0.9530
Clinical Trials	5.2%	8.2%	0.0181
Transplant	18.5%	27.7%	< 0.0001