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WILEY

BRIEF REPORT

# Racial and ethnic disparities in acuity of presentation among children with newly diagnosed acute leukemia

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These data were previously presented as an oral abstract at the American Society of Hematology Annual Meeting in December 2018 and published in *Blood* under the title "Increased disease burden among Black children compared to White children with newly diagnosed acute myeloid leukemia."

## Abstract

We evaluated disparities in disease burden, organ dysfunction, vital signs, and timing of therapy in children newly presenting with acute leukemia. Among 899 patients with acute leukemia diagnosed at two large children's hospitals, a priori lab-based definitions of high disease burden, infection risk, renal dysfunction, and coagulopathy were applied to electronic health record data. Black patients with acute myeloid leukemia had increased prevalence of elevated white blood cell count and uric acid; Black patients with acute lymphoblastic leukemia demonstrated increased prevalence of coagulopathy. Black patients' presentation more frequently included multiple lab abnormalities consistent with advanced physiologic dysfunction. No differences were found in days to therapy initiation.

**Abbreviations:** ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; CBC, complete blood count; CI, confidence interval; ED, emergency department; EHR, electronic health record; FPL, federal poverty level; ICU, intensive care unit; NDI, Neighborhood Disorganization Index; NH, non-Hispanic; PR, prevalence ratio; SES, socioeconomic status; WBC, white blood cell.

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**KEYWORDS**
diagnosis, disparities, pediatric leukemia, race

## 1 | INTRODUCTION

Racial and ethnic disparities in mortality among children with acute leukemia are only partially understood. Across acute leukemia types, the induction period of chemotherapy treatment carries the highest mortality.[1] Induction complications are substantially more common in patients with acute myeloid leukemia (AML) than acute lymphoblastic leukemia (ALL). We reported that children with AML requiring intensive care for multiorgan system failure at diagnosis have a 14-fold higher risk of dying during Induction I compared with those not requiring such intervention. Black patients are more likely to require intensive care unit (ICU)-level resources at initial presentation compared with White patients. After accounting for receipt of intensive care within the first 72 hours of initial admission, we found no racial differences in ensuing use of ICU-level resources during Induction,[2] or in subsequent chemotherapy.[3] This suggests that clinical status in the peri-diagnostic period drives disparities in early mortality and overall survival in AML. In contrast, despite persistence of racial and ethnic disparities in ALL survival, induction mortality disparities were not observed in recent cooperative group clinical trials.[4]

Using electronic health record (EHR) data from two large institutions, we examined racial, ethnic, and socioeconomic status (SES) differences in presenting clinical severity and management to assess potential contributions to survival disparities. We hypothesized that Black patients are more likely to both present with abnormal vital signs and have abnormal labs within the first 72 hours following admission. We further hypothesized that race and ethnicity do not affect timing to receipt of key interventions for diagnosis and initiation of chemotherapy.

## 2 | METHODS

Acute leukemia patients (aged 0–22 years) diagnosed between 2006 and 2014 at Children's Hospital of Philadelphia and Texas Children's Hospital were included. Patients transferring from a referring hospital or with race specified as Asian, other, or unknown were excluded due to small sample sizes. Age, sex, self-reported race and ethnicity, residential address at diagnosis, and results of selected laboratory tests from emergency department (ED) presentation through 72 hours were extracted from the EHR via an automated approach.[5] The first set of vital signs (blood pressure, heart rate, respiratory rate, and temperature) and timing of key diagnostic evaluations were manually abstracted from the EHR. Institutional review board (IRB) approval was obtained at each site.

Patients were classified into three mutually exclusive categories that combined self-reported race and ethnicity: Hispanic (any race), non-Hispanic White, and non-Hispanic Black/African American (hereafter referred to as Black). Patient-level residential addresses were geocoded and linked to census-derived block group. Block group SES was assessed by the following measures: median household income (less than the US federal poverty level [FPL] of $24,300 for a family of four in 2016 vs. >FPL), unemployment (cohort-based quartiles), and the Neighborhood Disorganization Index (NDI), a metric based on proportions of unemployment, households receiving public assistance, persons below the FPL, adults without a high school diploma, female-headed households, renter-occupied residences, and residential tenure less than 1 year.[6] Details of the process for construction of the NDI are included in Table S6. Vital signs were interpreted using age-specific American Heart Association reference ranges. Composite lab-based definitions created a priori based on clinical experience were used to identify high leukemia disease burden (white blood cell [WBC] count > 50 or uric acid > 10), risk of infection (absolute neutrophil count [ANC] < 200 cells/$\mu$L), renal dysfunction (creatinine > 1.0 IU/L), abnormal coagulation (prothrombin time > 17 seconds or fibrinogen < 100 mg/dL) and hepatic dysfunction (AST/ALT [aspartate transaminase/alanine transaminase] > 200 IU/L or bilirubin > 2.5 mg/dL or GGT > 150 IU/L) (Table S1). For cases with multiple results in 72 hours, the most abnormal lab result was used. We captured time (in minutes) from presentation to ED to first complete blood count (CBC), first lumbar puncture, first bone marrow aspirate/biopsy, and first dose of parenteral chemotherapy during the diagnostic inpatient admission.

Log-binomial regression was used to compute unadjusted prevalence ratios (PR) and corresponding 95% confidence intervals (CI) comparing laboratory abnormalities by race and ethnicity (non-Hispanic White patients as reference group). Stratification by leukemia type (AML vs. ALL) allowed for assessment of heterogeneity by disease. Covariates associated with exposures (defined by >10% change in point estimate) were included in multivariable models to obtain adjusted PRs for laboratory abnormalities by race and ethnicity. Median times to key interventions were compared by race and ethnicity using the Kruskal–Wallis test (also called the one-way analysis of variance on ranks). A $p$-value less than .05 was considered statistically significant.

## 3 | RESULTS

The cohort included 899 (474 non-Hispanic White, 318 Hispanic, and 107 Black) patients with acute leukemia (Table 1). The majority were male and between ages 1 and 9 years. Nearly all (94%) lived in block groups with a median household income above the FPL.

**TABLE 1** Patient characteristics stratified by acute leukemia type.

| Characteristics | AML | | | | | ALL | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Total (*N*) | Non-Hispanic White (%) | Non-Hispanic Black (%) | Hispanic (%) | *p* | Total (*N*) | Non-Hispanic White (%) | Non-Hispanic Black (%) | Hispanic (%) | *p* |
| Age (years) | | | | | .61 | | | | | .23 |
| <1 | 20 | 15 | 10 | 8.6 | | 23 | 2.2 | 5.1 | 2.8 | |
| 1–9 | 64 | 41 | 45 | 46 | | 586 | 71 | 56 | 72 | |
| 10–18 | 61 | 42 | 38 | 46 | | 221 | 26 | 37 | 24 | |
| >18 | 3 | 1.4 | 6.9 | 0 | | 10 | 1.5 | 1.3 | 0.4 | |
| Sex | | | | | .80 | | | | | .34 |
| Male | 74 | 51 | 48 | 46 | | 467 | 57 | 62 | 54 | |
| Female | 74 | 49 | 52 | 54 | | 373 | 43 | 38 | 46 | |
| Site | | | | | <.0001 | | | | | <.0001 |
| CHOP | 77 | 70 | 66 | 11 | | 379 | 63 | 56 | 15 | |
| TCH | 71 | 30 | 35 | 89 | | 461 | 37 | 44 | 85 | |
| Weight status | | | | | .007 | | | | | <.0001 |
| Underweight | 10 | 4.4 | 18 | 0 | | 58 | 6.3 | 11 | 5.9 | |
| Normal for age | 88 | 72 | 46 | 56 | | 517 | 71 | 53 | 54 | |
| Overweight | 18 | 10 | 25 | 13 | | 119 | 13 | 17 | 15 | |
| Obese | 26 | 13 | 11 | 31 | | 126 | 9.1 | 19 | 25 | |
| AML risk group | | | | | .21 | | | | | |
| High | 33 | 46 | 35 | 20 | | | | | | |
| Intermediate | 23 | 26 | 20 | 28 | | | | | | |
| Low | 91 | 28 | 45 | 52 | | | | | | |
| ALL phenotype | | | | | | | | | | .01 |
| B-cell ALL | | | | | | 676 | 88 | 79 | 92 | |
| T-cell ALL | | | | | | 88 | 12 | 21 | 8 | |
| CNS status | | | | | .37 | | | | | .02 |
| CNS 1 | 79 | 67 | 48 | 51 | | 506 | 66 | 54 | 71 | |
| CNS 2 and 3 | 55 | 33 | 52 | 49 | | 258 | 34 | 46 | 29 | |
| Years | | | | | .24 | | | | | <.0001 |
| 2006–2010 | 65 | 36 | 12 | 17 | | 396 | 56 | 46 | 48 | |
| 2011–2014 | 70 | 35 | 17 | 18 | | 460 | 44 | 54 | 52 | |
| Block level neighborhood disorganization index | | | | | <.0001 | | | | | <.0001 |
| Q1 <0.53 | 176 | 37 | 11 | 9 | | 34 | 41 | 7 | 9 | |
| Q2 0.53–0.93 | 203 | 36 | 16 | 18 | | 29 | 24 | 14 | 24 | |
| Q3 0.93–1.53 | 178 | 20 | 20 | 31 | | 41 | 24 | 45 | 32 | |
| Q4 >1.53 | 189 | 7 | 53 | 43 | | 29 | 10 | 35 | 35 | |
| Block level median household income | | | | | .39 | | | | | .0001 |
| <FPL | 8 | 4.2 | 14 | 2.9 | | 46 | 1.8 | 14 | 8.5 | |
| >FPL | 139 | 95 | 86 | 97 | | 791 | 98 | 86 | 91 | |

(Continues)

**TABLE 1** (Continued)

| Characteristics | AML Total (*N*) | AML Non-Hispanic White (%) | AML Non-Hispanic Black (%) | AML Hispanic (%) | AML *p* | ALL Total (*N*) | ALL Non-Hispanic White (%) | ALL Non-Hispanic Black (%) | ALL Hispanic (%) | ALL *p* |
|---|---|---|---|---|---|---|---|---|---|---|
| Block level unemployment | | | | | <.0001 | | | | | .06 |
| Q1 <2.5% | 179 | 27 | 18 | 20 | | 31 | 28 | 17 | 18 | |
| Q2 2.5%–4.3% | 180 | 26 | 13 | 23 | | 35 | 32 | 10 | 26 | |
| Q3 4.3%–7.2% | 198 | 26 | 21 | 27 | | 37 | 25 | 34 | 27 | |
| Q4 >7.2% | 204 | 20 | 49 | 30 | | 31 | 14 | 38 | 29 | |

Abbreviations: ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; CHOP, Children's Hospital of Philadelphia; CNS, central nervous system; FPL, federal poverty level; TCH, Texas Children's Hospital.

**TABLE 2A** Prevalence ratio of laboratory abnormalities by race and ethnicity.

| Lab | AML NH White Prev (%) | AML NH Black Adjusted PR[a] [95% CI] | AML Hispanic Adjusted PR[a] [95% CI] | ALL NH White Prev (%) | ALL NH Black Adjusted PR[b] [95% CI] | ALL Hispanic Adjusted PR[b] [95% CI] |
|---|---|---|---|---|---|---|
| Total *N* | 71 | 29 | 35 | 403 | 78 | 283 |
| WBC >50 | 25% | **2.46 [1.10–5.50]** | 1.84 [0.69–4.86] | 18% | 1.14 [0.77–1.68] | 1.16 [0.82–1.62] |
| ANC <200 | 32% | 0.92 [0.38–1.75] | 0.91 [0.46–1.82] | 40% | 0.68 [0.46–1.01] | 0.98 [0.80–1.21] |
| Uric acid >10 | 2% | **2.37 [1.33–4.16]** | 0.98 [0.56–1.69] | 7% | 1.15 [0.62–2.11] | 0.87 [0.47–1.60] |
| Coagulopathy | 24% | 0.61 [0.17–2.20] | 0.77 [0.13–4.60] | 6% | **2.07 [1.04–4.10]** | 0.75 [0.21–2.68] |
| Abnormal LFTs | 5% | 2.26 [0.41–12.51] | 1.10 [0.08–14.76] | 6% | 1.87 [0.88–3.98] | 1.46 [0.82–2.60] |
| Creatinine >1.0 | 9% | 2.60 [0.65–10.40] | 3.45 [0.82–14.42] | 5% | 1.07 [0.46–2.54] | 0.63 [0.26–1.49] |
| >1 Lab abnormalities | 13% | 2.16 [0.64–7.34] | 1.65 [0.25–11.15] | 6% | **2.20 [1.15–4.23]** | 1.14 [0.57–2.26] |

Abbreviations: ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; ANC, absolute neutrophil count; CBC, complete blood count; CI, confidence interval; LFT, liver function test; NH, non-Hispanic; PR, prevalence ratio; WBC, white blood cell.

[a]Prevalence ratios for AML are adjusted for institution, body mass index (BMI), and AML risk group.

[b]Prevalence ratios for ALL are adjusted for age, institution, BMI, median income, year, central nervous system (CNS) status, and ALL phenotype.

Vital signs at presentation were similar across racial and ethnic groups (Tables S2A and S2B). Black patients with AML had an increased prevalence of elevated WBC count (PR 2.46; 95% CI: 1.10–5.50) and elevated uric acid (PR 2.37; 95% CI: 1.33–4.16) at presentation (Table 2A). Black patients with ALL demonstrated increased prevalence of coagulopathy (PR 2.07; 95% CI: 1.04–4.10). Black patients with AML or ALL were also more likely to have two or more laboratory abnormalities (PR 2.16; 95% CI: 0.64–7.34, and PR 2.20; 95% CI: 1.15–4.23, respectively). There were suggestions of increased prevalence of hepatic and renal dysfunction among Black patients with AML (PR 2.26; 95% CI: 0.41–12.51 and PR 2.60; 95% CI: 0.65–10.40, respectively). Hispanic patients had no statistically significant differences in laboratory findings; however, among those Hispanic patients with AML, renal dysfunction was more prevalent (PR 3.45; 95% CI: 0.82–14.42).

Across all groups, median time to CBC was less than 2 hours, to bone marrow biopsy and lumbar puncture was 25—38 hours, and to induction chemotherapy was 2—3 days (Table 2B). Black and Hispanic patients had longer median times to all diagnostic evaluations compared to the non-Hispanic White population, with statistically significant differences in the time to bone marrow evaluation and lumbar puncture. Neighborhood SES was not significantly associated with primary or secondary outcomes in either AML or ALL (Tables S3–S6).

## 4 | DISCUSSION

Using EHR data from two large, diverse, and relatively representative academic cancer centers in the United States, we demonstrated that high disease burden at presentation of newly diagnosed AML is more prevalent in Black children than non-Hispanic White children. We also find that coagulopathy is more prevalent in Black patients with ALL. Black patients have a greater than two-fold risk of presenting with substantial dysfunction in multiple systems across acute leukemia types. Importantly, fewer statistically significant differences in presenting laboratory results were detected between Hispanic and non-Hispanic White patients.

**TABLE 2B** Median time (and time range) to key diagnostic testing and treatment by race and ethnicity.

| | NH White | NH Black | Hispanic | *p* |
|---|---|---|---|---|
| Hours to CBC | 1.7 [−0.9, 55] | 1.8 [0, 10] | 1.8 [−0.8, 24] | .06 |
| Hours to bone marrow evaluation | 24.8 [0.8, 233] | 26.6 [0.5, 206] | 30.2 [0.8, 208] | .01 |
| Hours to lumbar puncture | 27.8 [0.5, 312] | 34.7 [10.3, 231] | 38.0 [4.2, 474] | .04 |
| Days to chemotherapy | 2.0 [0, 13] | 2.0 [1, 11] | 2.0 [0, 12] | .96 |

Abbreviations: CBC, complete blood count; NH, non-Hispanic.

High presenting WBC counts are associated with tumor lysis syndrome, hyperuricemia, and acute kidney injury, as well as with early mortality through acute pulmonary or central nervous system (CNS) hemorrhage.[7,8] Thus, the higher frequency of ICU-level care at diagnosis among Black patients with AML[2] may be partially explained by high leukemic burden at diagnosis.

Higher prevalence of elevated presenting WBC counts may be attributable to disease biology or delayed presentation, or both. Previous studies suggest monocytic AML subtypes (including those with *KMT2A* rearrangements) present more frequently with elevated WBC count.[8] While *KMT2A*-rearranged AML is not broadly associated with Black race, the specific *KMT2A*-rearrangement t(6;11)(q27;q23) that carries a poor prognosis is more common among Black children.[9] In contrast to our findings, in a previous study a difference in median WBC was not found between Black and White children.[10] The lack of difference in that study may be due to selection bias caused by using trial-collected biospecimens, which favors children with lower acuity at presentation.[11]

Access to care is another potential contributor to disparate presentations. We have previously shown that financial disincentives associated with seeking emergency care delay presentation for medical attention despite symptom burden.[12] We evaluated time to receipt of key evaluations, and found no difference in time to initiation of chemotherapy, although there were statistically significant differences in the time to bone marrow evaluation and lumbar puncture among non-Hispanic White patients compared to Black and Hispanic patients. This is consistent with data from adult AML showing no differences in time to referral for leukemia treatment.[13]

We recognize limitations in interpreting our findings. Although a strength of our approach was inclusion of patients treated on and off clinical trials, there was limited power to detect statistically significant differences, particularly for rare outcomes and rare populations. As some analyses were unadjusted, there is risk of uncontrolled confounding. The thresholds for laboratory values used as proxies for severity of leukemia presentation were chosen a priori based solely on clinical experience. Individual-level SES and insurance data were not available. Neighborhood measures of SES do not fully capture the impact of residential segregation and neighborhood deprivation, which would more effectively assess the impacts of structural racism. We were unable to evaluate structural barriers that patients faced prior to presentation to the treating hospital.

The concentration of disparities in AML may be related to the prevalence of presentation acuity and early mortality relative to ALL. These data generate additional questions and hypotheses; for example, could strengthening supportive care immediately following diagnosis prevent complications associated with high disease burden? Another approach could focus on improving upstream referral pathways and limiting insurance barriers to allow for earlier intervention prior to the development of organ dysfunction. Future studies should focus on determining if these findings hold true in larger cohorts of diverse patients treated on and off clinical trials, including adults with acute leukemia. In addition, identifying drivers of high disease burden (whether social or biologic) and testing potential interventions to limit the effects of high disease burden in Black patients with AML are essential next steps.

## AUTHOR CONTRIBUTIONS

Kelly D. Getz, Michael E. Scheurer, M. Monica Gramatges, Karen R. Rabin, and Richard Aplenc provided patients to the cohort. Kelly D. Getz, Yimei Li, Evanette Burrows, Vicky Tam, and Tamara P. Miller extracted and analyzed data. Lena E. Winestone wrote the draft manuscript. All authors provided critical review of the manuscript and approved the final manuscript.

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## CONFLICT OF INTEREST STATEMENT

Brian T. Fisher serves on a data safety monitoring board for Astellas. His institution also receives funding to support research he performs on behalf of Pfizer, Merck, and Allovir. The remaining authors declare no potential conflicts of interest.

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the senior author. The data are not publicly available due to privacy or ethical restrictions.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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