The 65th ASH Annual Meeting Abstracts

POSTER ABSTRACTS

906.OUTCOMES RESEARCH-MYELOID MALIGNANCIES

The Impact of Air Pollutants on a Chicagoland Cohort of Patients with Acute Myeloid Leukemia

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Background

Outcomes in acute myeloid leukemia (AML) can be impacted by cytogenetic/molecular features along with environmental factors such as previous exposure to cytotoxic therapy and county-level income. Our group previously identified structural racism, defined as systemic disadvantage experienced by certain groups of people, as a mediator of disparities within a cohort of 822 patients (pts) diagnosed with AML in the Chicagoland area even when accounting for genetic features of disease (Abraham et al, Blood 2022). Air pollution disproportionately impacts racial-ethnic minorities and has been demonstrated to impact DNA methylation patterns in pts which in turn influence disease biology (Tessum et al, Sci Advances 2021; Rider et al, Clin Epigenetics 2019). With that in mind we aimed to investigate the impact of air pollutant exposures that have been associated with both leukemia development/mortality and with air pollution disparities in historically redlined cities.

Methods

Adult AML (non-APL) pts diagnosed between 2012 and 2018 at six academic cancer centers in the Chicago area were included. Pts' census tracts were joined with measures of pollution obtained from the Center for Disease Control National Environmental Public Health Tracking Network and the 2014 National Air Toxics Assessment: 1,3-butadiene, benzene, diesel particulate matter (PM), polycyclic aromatic hydrocarbons and polycyclic organic matter (PAHPOM), and PM 2.5 μm (2014 averaged daily census tract measures). Pollutant exposures by racial/ethnic groups were determined using a Kruskal-Wallis rank sum test and pairwise Wilcoxon rank sum tests. Univariate logistic regressions for each pollutant were performed for 2010 European Leukemia Net (ELN) AML criteria and *TP53* mutation status. Survival among pts in the most and least polluted census block quartiles for each pollutant was compared using Kaplan-Meier survival analysis.

Results

Our multi-center database contained 789 pts with AML with census tract data, allowing for air pollutant data collection. Median age of diagnosis was 62 years old. In addition, 59.9% of pts were non-Hispanic White (NHW), 15.3% were non-Hispanic Black (NHB), and 15% of pts were Hispanic. Additional characteristics are summarized in **Table 1.** There was significantly higher

median exposure to 1,3-butadiene, benzene, diesel PM, PAHPOM, and PM2.5 in NHB and Hispanic pts when compared to NHW pts. Hispanic pts had significantly median higher exposure to 1,3-butadiene and benzene when compared to NHB pts while NHB pts had significantly higher median exposure to PM2.5 (**Table 2**). With univariate analysis, no pollutant exposure was found to be significantly associated with ELN 2010 risk category or with *TP53* mutation status. We also compared median OS (mOS) from AML diagnosis between the quartile with the lowest exposure (Q1) and quartile with the highest exposure (Q4). For 1,3-butadiene mOS was 2.52 years (Q1) vs. 2.96 years (Q4) (p=0.75), for benzene it was 2.65 years vs 3.0 years (p=0.38), for diesel PM it was 2.56 years vs 2.96 years (p=0.56), for PAHPOM it was 2.76 years vs. 2.96 years (p=0.31), and for PM2.5 it was 2.92 years vs. 2.70 years (p=0.32).

Conclusions

We analyzed the impact of 1,3-butadiene, benzene, diesel PM, PAHPOM, and PM2.5 on outcomes in pts with AML in the Chicagoland area. While there were significant differences in median exposure to pollutants by race/ethnicity, there was no significant association between pollutant exposure and ELN 2010 risk category or TP53 mutation analysis. We also analyzed overall survival between Q1 and Q4 of exposure for each pollutant and found no significant difference. While pollutant exposure disproportionately impacts racial-ethnic minorities, individual pollutants did not appear to be a major driver of outcomes in AML. Limitations include the source of our air pollutant data, where misclassification of pollutants may exist and impact the ability to detect true associations.

Table 1: Patient demographics and disease characteristics

Age at AML Diagnosis	n=789
Median age (range)	62 (18-95)
Age 18-59, n (%)	350 (44%)
Age 60-74, n (%)	313 (40%)
Age 75+, n (%)	125 (16%)
Race/Ethnicity, n (%)	n=789
Non-Hispanic White	473 (59.9)
Non-Hispanic Black	121 (15.3)
Hispanic	118 (15.0)
Non-Hispanic Other	77 (9.8)
ELN 2010 Categorization, n (%)	n=750
Favorable	112 (14.9)
Intermediate-I	280 (37.3)
Intermediate-II	136 (18.1)
Adverse	222 (29.6)
TP53 Mutation Status, n (%)	n=512
Mutated – pathogenic	43 (8.4)
Unmutated/mutated-non known pathogenic	469 (91.6)

Table 2: Median Pollutant Exposure by Race/Ethnicity

Pollutant	Non-Hispanic White	Non-Hispanic Black	Hispanic
1,3-butadiene	0.047888	0.05674*	0.0627515*#
Benzene	0.564339	0.622442*	0.672733*#
Diesel PM	0.657941	0.832843*	0.9168055*
PAHPOM	0.002269	0.00261*	0.002719*
PM 2.5	12.52878	13.13635*$	13.01355*

*Median exposure significantly higher when compared to non-Hispanic White
#Median exposure significantly higher when compared to non-Hispanic Black
$Median exposure significantly higher when compared to Hispanic

Figure 1

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