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**“Health Disparities in High-Risk Lung Cancer Families and Their Association with Smoking, Environmental Exposures, and Other Etiological Factors”**

Lung cancer (LC) is by far the leading cause of cancer-related deaths worldwide. LC survival has only improved marginally over the last decades with the five-year survival rate for LC being lower than most other leading cancer sites. African Americans (AAs) have a higher incidence rate and lower survival rate for LC in comparison to all other racial and ethnic groups in the United States. In addition, incidence rates among AAs and European Americans (EAs) vary with histology of LC. Although tobacco smoking has been identified as the major risk factor for LC, studies have shown there is a genetic component involved in the development of the disease. About 25% of LC cases have at least one first- or second-degree relative, indicating that family history is a relevant risk factor. The goal of this study is to characterize genetic, clinical, and environmental risk factors among individuals of EA and AA ancestry from the high-risk families with LC that could hold important clinical value to address LC disease disparity.

Study participants with LC were recruited from a network of 30 hospitals from Louisiana along with multiple states across the country. Study participants with at least two confirmed cases of primary LC within the family were eligible. Participants were divided into two subgroups: Familial ( $\geq 2$  LC cases/family) and Hereditary LC (HLC families) ( $\geq 3$  affected LC cases/ family). Medical and pathology reports were obtained from hospitals along with demographic and environmental data from the families. A total of 192 study participants (157 EA and 35 AA) from both familial and HLC families from the years 1992 through 2021 were used in this study. Data abstracted from the pathology, clinical reports, and study questionnaire was entered into spreadsheets and analyzed. Histology of LC diagnosis and clinical reports on mutation analysis were documented.

The preliminary analyses of results have found that the average age of onset for AAs is significantly lower than in EA (P value < 0.0001). Additionally, while smoking is commonly referred to as a major contributor to LC disparities, AAs were found to have significantly lower pack years of cigarette use than EAs (P value < 0.05). The average age the AA participants 'begin to smoke' was also found to be significantly lower than EAs (P value < 0.05). The majority of the study participants with LC were diagnosed with adenocarcinoma irrespective of the number of pack-years for cigarette use. Mutation analysis in the clinical report for a small number of study participants in the EA families provided limited information.

Additional analysis is ongoing. Clinical and pathological characterization in association with risk factors from high-risk families with EA and AA ancestry will provide us with a better understanding behind the disproportionate distribution of incidence and survival for LC in the AA population.