The Connection Between the Development of Acute Myeloid Leukemia and Benzene Uptake

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Research into understanding the effects of how benzene intake can lead to the development of Acute Myeloid Leukemia (AML) is being overlooked in many developing countries. These countries continue to use benzene as a source of raw materials in plastic and detergents without the proper protection or following laws from the Occupational Safety and Health Administration. AML has an estimated 20,000 cases per year in the United States alone and is a serious type of cancer with 11,000 US annual deaths. Because of benzene's unique chemical characteristics, once broken down in the body, it allows the reactivation of monoaldehydes which are electrophiles that can readily react with proteins and peptides in the long noncoding cells RNA -OBFC2A (LncRNA-OBFC2A) signaling pathway. The lncRNA-OBFC2A pathway allows the regulation of gene transcription by modulating DNA histones. Here, I present a review of clinical studies collected from patients with different levels of exposure to benzene and discuss how this affected the overall function of cells in their bodies. Literature on benzene exposure shows that working for five years in an environment with 0.016-0.413 parts per million (ppm) of benzene is correlated with higher chances of developing acute myeloid leukemia compared to those that have been exposed to 0.1 ppm over the same period. This exposure is especially seen in electronic factories in developing countries using higher benzene ppm levels. Understanding the effects of benzene on cell function, and collecting data comparing AML and electronic workers who are exposed to these conditions will allow data that will narrow down at which ppm exposure levels would inhibit the lncRNA-OBFC2A pathway.