Identifying the Relationship Between Benzene Exposure and the Development of Acute Myeloid Leukemia

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AIM and OBJECTIVES
To examine the mechanistic relationship between benzene exposure and acute myeloid leukemia (AML).
• To describe the normal metabolic pathways and cellular proliferation in hematopoietic stem cells (HSC).
• To outline the abnormal cellular processes that occur in AML.
• To describe the biomechanic pathways by which benzene exposure leads to AML, including oxidative stress and the dose response relationship.
• To identify areas of needed research.

METHODOLOGY
• Conducted a systematic search of the scientific literature written in English and published in the past five years (2006-2012) using online databases and professional websites.
• Reviewed select articles and summarized findings.
• Created a poster.
• Outlined an article for submission to a peer-review journal.

BACKGROUND
• AML is a rare and highly malignant cancer.
• It is estimated 13,780 new cases of AML will be identified in the United States this year with a 74% mortality rate.
• AML develops from alterations in the survival and proliferation of HSCs in the bone marrow microenvironment.
• If AML is not diagnosed early and aggressively treated, bone marrow failure and death occur.
• Less than 10% of AML cases are caused by genetic factors, and up to 70% are of unknown origin.
• Twenty percent of AML cases have known environmentally-related etiologies.
• Environmental risk factors include cigarette smoke, radiation exposure, and exposure to chemicals such as benzene.
• Benzene is one of the most commonly used chemicals in the U.S.
• Exposure to environmental benzene, even at levels lower than the Occupational Safety and Health Administration’s limit of 1 ppm, has been associated with AML.

FINDINGS
• Benzene and/or its metabolites induce AML via oxidative stress, aryl hydrocarbon receptor (AhR) dysregulation and reduced immunosurveillance.
• These processes lead to the generation of leukemic stem cells (LSC) and subsequent evolution to leukemia by:
  • Targeting critical genes and pathways through the induction of genetic, chromosomal or epigenetic abnormalities, and creating genomic instability in HSCs.
  • Creating stem cell niche and stromal cell dysregulation.
  • Inducing apoptosis of HSCs and stromal cells.
  • Altering proliferation and differentiation of HSCs.

AREAS of NEEDED RESEARCH
• Determine the dose response relationship of benzene on AML.
• Further examine the effects of benzene and its metabolites on HSCs, the stem cell niche, and stromal cells.
• Further identify the roles of multiple metabolites in benzene toxicity and the pathways that lead to their formation.
• Further assess health risks associated with benzene exposure, particularly at low levels.
• Identify additional causative factors associated with the development of AML.

NURSING IMPLICATIONS
• Environmental determinants of health and disease are widespread and vital to the assessment, diagnosis, intervention, planning, and evaluation in nursing practice.
• Nurses need to be aware of environmental factors that impact health and disease.
• Nurses need to identify environmental contaminants in order to eliminate and control health hazards.

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