Connection Between the Development of Acute Myeloid Leukemia and Benzene Uptake

Tyler Pham and Marc Nahmani
Division of Sciences & Mathematics, University of Washington | Tacoma, WA 98402

ABSTRACT

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.

METHODS

The strategy begins with the preparation of researching data on benzene and the overall effects on the human cells from primary and secondary articles. The first step is to gather an understanding of what benzene is and how the body metabolizes it. Knowing that the body metabolizes benzene, what type of effect this would have on a cell: would this cause n inhibition of a proton or reconfigure DNA/RNA? This would then correlate with Acute Myeloid Leukemia as the cells becoming inhibited are blood cells.

METABOLIC PATHWAYS FOR BENZENE REACTIVITY & CELL DAMAGE

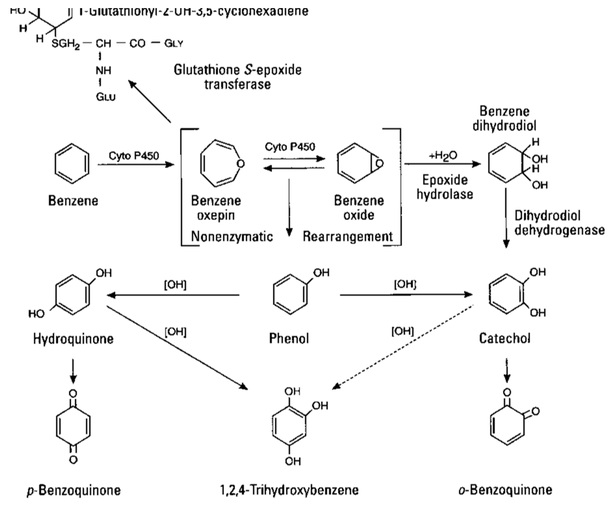

Figure 1. Mechanism of Benzene metabolism in the liver and bone marrow.

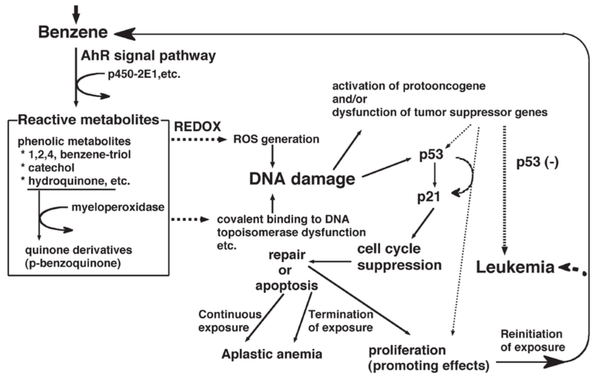

Figure 2. The possible benzene metabolism mechanism for inducing acute myeloid leukemia

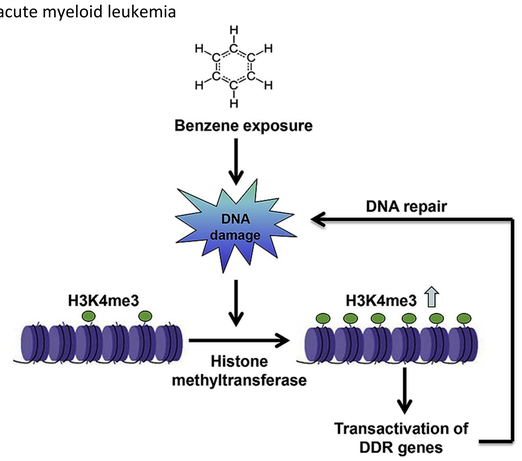

Figure 3. The pathway of how benzene that is metabolized attacks cells histones and what occurs

EFFECTS OF BENZENE ON CELL FUNCTION & HUMAN HEALTH OUTCOMES

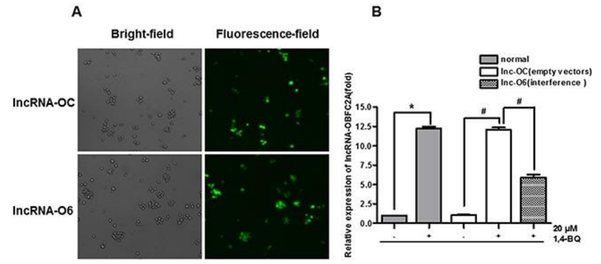

Figure 4. A0 AHH-1 cells that were transfected by lentivirus vectors with the lncRNA-OBFC2A for 72 hours B) the expression of lncRNA-OBFC2A in the AHH-1 cells was detected

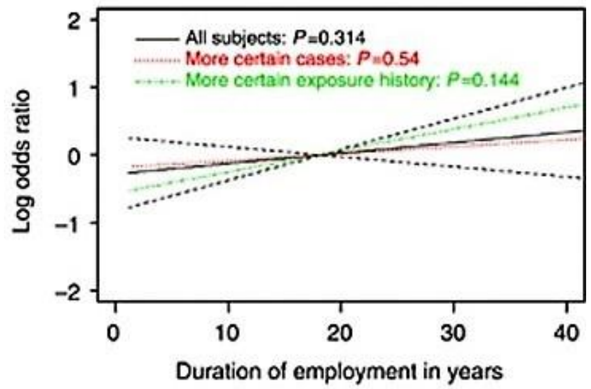

Figure 5. The comparison of a subject acquiring AML over a given amount of years being exposed to Benzene

Characteristic	Benzene Exposure N[1] = 74	No Benzene Exposure N[1] = 337	OR (95% CI)
Sex N (%)			
Female	16 (22)	152 (45)	Ref.
Male	58 (78)	185 (55)	**3.00 (1.64–5.49)**
Age at diagnosis N (%)			
≤ 50	18 (24)	101 (30)	0.69 (0.17–2.83)
50 – 59	17 (23)	72 (21)	0.92 (0.40–2.10)
60 – 69	28 (38)	102 (30)	Ref.
70 – 79	11 (15)	62 (18)	0.63 (0.26–1.54)
≥ 80	-	-	
Education N (%)			
=< HS	23 (32)	98 (29)	Ref.
Post-HS	35 (48)	121 (36)	1.24 (0.69–2.25)
College graduate	15 (21)	116 (35)	0.56 (0.28–1.14)
Smoking Status[2] N (%)			
Never	20 (28)	154 (46)	Ref.
Former	37 (52)	122 (37)	2.35 (1.27–4.34)
Current	15 (21)	57 (17)	2.02 (0.97–4.23)
Lived or worked on a farm ≥ 1 year N (%)	29(39)	125 (37)	1.05 (0.62–1.78)
No	45 (61)	211 (63)	Ref.
Personal History of cancer N (%)	17 (23)	50 (15)	1.68 (0.89–3.17)
No	57 (77)	287 (85)	Ref.
Cytogenetics			
Abnormal	44 (63)	176 (58)	1.27 (0.73–2.21)
Normal	26 (37)	127 (42)	Ref.

Figure 6. The comparison of demographic and characteristics in benzene exposed and unexposed cases

CONCLUSIONS

- Strong correlation between 0.016-0.413 ppm benzene to the development of Acute myeloid leukemia
- Strong correlation between oil refinery and terminal workers to develop Acute myeloid leukemia
- Critical to understand the relationship of lncRNA-OBFC2A and NOTCH1 when inhibited

FUTURE DIRECTIONS

- Revisit and allow punishment of factories in countries that simply overlooks this problem and allows the use of benzene as a method to form products
- More research is needed to understand the mechanistic pathway of how benzene can induce acute myeloid leukemia

Acknowledgements

Special thanks to:

- Dr. Marc Nahmani for organizing this class and keeping me accountable during the time of my project
- My peers for providing insights throughout the process
- UW Tacoma TLC for helping me with my research

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