Cleveland's Center for Healing
HYPERION
Functional Medicine

BLADDER CANCERS

There are three routes of entry for pesticides and other toxins into the human body including the skin and/or eyes, by inhalation, and through ingestion of food and liquids containing toxins. Exposure to toxins in bladder cancer is through ingestion of toxic foods and from toxin exposures in the environment. One possible mode of action potentiating bladder cancer risk by pesticide exposure is by the "urogenous contact" hypothesis: active carcinogens created via pesticide metabolites dissolved in urine can influence cells of the bladder tissue[1].

Mitochondrial mutations in bladder cancer tend to be induced by damage from tobacco smoking, ionizing radiation, ozone, pesticides, and heavy metals[2]. More than a dozen chemicals and industrial agents have been linked to the development of bladder cancer. From Water pollution: contaminants include inorganic arsenic, disinfection byproducts formed during water treatment such as chlorine, and nitrate. Well water contains many contaminants due to runoff in the soil from farms and industry. In industry, exposure to toxins in rubber manufacturing, transportation, dry cleaning, aluminum production, metalworking and machining, textile and dye manufacturing, painting and hair salons have an increased risk of causing bladder cancer development. The higher risk is linked to exposures to aromatic amines, perchloroethylene, mineral oils/ metal working fluids, polycyclic aromatic hydrocarbons (PAHs), diesel exhaust and coal-tar pitch. Air pollution also plays a role: Based on evidence from studies of workers exposed to toxins, various components of air pollution, including diesel exhaust and PAHs from vehicles and industries have been shown to contribute to bladder cancer[3]. A recent study published in 2023 found that heavy metals and trace elements contributed to urinary bladder cancer. Significantly higher levels of arsenic, manganese, copper, selenium, cadmium, lead, and mercury were found in patients with urinary bladder cancer[4].

Tumor cells in bladder cancer are assisted by mitochondria that have been damaged leading to reprogramming of their functions. This reprogramming increases energy needed for bladder and other types of cancer cells by switching to a specialized form of metabolism known as the "Warburg Effect" for growing the tumor. These results of reprogramming favor tumor progression, blood vessel growth for the tumor, and suppression of the immune system.

Through elimination toxins, mitochondrial damage will be decreased.

Toxins and their effects are compounded over time through multiple exposures. Everyone has a history of toxin exposures and consequently plans need to be implemented to optimize avoiding exposures impacting this route of cancer and chronic disease. Optimizing detoxification in patients with cancer is a priority and a detoxification plan is put into place for all bladder cancer patients at the initial appointment with Hyperion functional medicine.

Hyperion functional medicine is positioned and offers to test for specific levels of heavy metals and toxins in patients in the functional oncology program for bladder cancer.

In partnership with healingstrong

[1]Lucchesi CA, Vasilatis DM, Mantrala S, Chandrasekar T, Mudryj M, Ghosh PM. Pesticides and Bladder Cancer: Mechanisms Leading to Anti-Cancer Drug Chemoresistance and New Chemosensitization Strategies. Int J Mol Sci. 2023 Jul 13;24(14):11395. doi: 10.3390/ijms241411395. PMID: 37511154; PMCID: PMC10380322.
[2]Huang CC, Liu HY, Hsu TW, Lee WC. Updates on the Pivotal Roles of Mitochondria in Urothelial Carcinoma. Biomedicines. 2022 Oct 1;10(10):2453. doi: 10.3390/biomedicines10102453. PMID: 36289714; PMCID: PMC9599371.
[3](https://bcan.org/wp-content/uploads/2019/05/Environmental-Risk-Factors.pdf).
[4]Panaiyadiyan S, Quadri JA, Nayak B, Pandit S, Singh P, Sachan A, Kaushal S, Sarwar S, Seth A, Shariff A. Association of heavy metals and trace elements in carcinoma urinary bladder: A case-controlled study. Indian J Urol. 2023 Jul-Sep;39(3):236-240. doi: 10.4103/iju.iju_143_23. Epub 2023 Jun 30. PMID: 37575163; PMCID: PMC10419772.