Colorectal Cancer, Blood Inflammatory Indicators, and Ubiquitous Drinking Water Chemical Exposure

Kiran Rana*

Editorial office, European Journal of Clinical Oncology, India

*Corresponding Author**

Kiran Rana
Editorial office,
European Journal of Clinical Oncology,
India
E-mail: oncology@scholarlymed.com

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Abstract

Trihalomethanes (THMs) and nitrate are common contaminants in drinking water that are linked to a higher risk of colorectal cancer, however the exact mechanisms are unclear. We looked into the relationship between inflammatory indicators and exposure to THMs and nitrate in drinking water, as well as the risk of colorectal cancer

Keywords: Drinking water • Trihalomethanes • Nitrate • Inflammation • Immune response • Colorectal cancer

Introduction

One of the most prevalent malignancies in both men and women, colorectal cancer accounts for 10% of all cancer cases worldwide and is on the rise in the majority of developed nations. With 1.2 million new cases and 10% of all malignancies in 2020, colorectal cancer was the third most prevalent malignancy. Almost half of all new instances take place in Europe and the Americas, and more than 65% of new cases happen in nations with high or very high levels of human development. Food is the primary causative element, and proven risk variables include total energy intake, consumption of red and processed meat, alcohol, as well as inactivity, body and abdominal fat, and adult height. Dietary fibre and a high intake of fruits and vegetables are protective factors. Colorectal cancer risk factors have included environmental exposures including nitrate in drinking water and Disinfection By-Products (DBPs). Disinfectants like chlorine react with the organic matter that is naturally present in raw water to produce disinfection Byproducts (DBPs), which are produced during the treatment of Drinking water. Trihalomethanes (THMs), which are often the most common and have been employed as indicators of DBP exposure in epidemiological research, make up the complex mixture of over 700 compounds that make up DBPs. Long-term exposure to THMs has been associated to colorectal cancer and can occur by ingestion, inhalation, and skin contact. For the greatest vs. lowest DBP exposure categories, the combined relative risk (RR) was calculated in a meta-analysis of 13 studies to be 1.27 (95% CI 1.08-1.50) for colon cancer and 1.30 (95% CI 1.06-1.59) for rectal cancer, with somewhat higher RR for case-control vs. cohort studies. There was no conclusive evidence of a link between lifetime total THM exposure and colorectal cancer in a multicase-control study done in Spain between 2008 and 2013 that included 2,047 colorectal cancer cases and 3,718 controls (Villanueva et al., 2017).

However, a negative correlation with chloroform and a positive association with brominated THMs were discovered, indicating that some THMs differ from others. In 2019, Jones et al. reported a connection between rectal cancer risk and exposure estimates of ingested total THM, bromodichloromethane, and trichloroacetic acid, but not for colon cancer (hazard ratioQ5vsQ1=1.71, 95% Confidence Interval (CI) 1.00-2.92). In conclusion, there is strong evidence linking DBP exposure to an increased risk of colorectal cancer, but heterogeneous associations for different exposures, populations, cancer sites, effect modifiers, and lack of knowledge of the underlying biological mechanisms prevent drawing a conclusion that links the two. Nitrate is a chemical that is frequently found in drinking water and is produced by fertilisers, industrial farming, and sewage. If endogenous nitrosation is present, ingested nitrate is likely a human carcinogen. Several studies have found higher risks at exposure levels below the regulatory limits for colorectal cancer, which has been repeatedly associated to long-term exposure to nitrate in drinking water. In a multicase-control study conducted in Spain between 2008 and 2013, 1,869 cases and 3,530 controls were identified, and the odds ratio (OR) of colorectal cancer for long-term exposure to >10 vs. 5 mg/day of nitrate was 1.49 (95% CI 1.24-1.78). However Jones et al. (2019) found no link between drinking water nitrate intake and the incidence of colon or rectal cancer in the Iowa Women's Health Study cohort (Jones et al., 2019). Moreover, Jones et al. (2019) were the first to look into how exposure to DBPs and nitrate in drinking water may affect the risk of developing colon and rectal cancer. They found no evidence that total THMs and nitrate interact to affect the risk of either cancer site. Colorectal cancer is a diverse illness that develops by a variety of sequences of events, including chronic inflammation, and inflammation is a crucial component of carcinogenesis. The immune system's reaction to an infection, an injury, or toxic exposures is inflammation. The immune system's cells create cytokines, a large class of tiny proteins important in cell signalling, among other things. THMs, and particularly chloroform, bromodichloromethane, dibromochloromethane, and bromoform, have been demonstrated to subchronically expose rodents to a dose-dependent reduction in cell-mediated immunity. After exposure to a variety of DBPs, a recent global transcriptional investigation in human intestinal epithelial cells revealed considerable effects on transcription genes associated to immunity and inflammation. Our findings imply that the negative health effects of DBP exposure may be significantly influenced by changes to genes linked to immunological and inflammatory pathways. During short-term DBP exposure in swimming pools, alterations in serum immunological indicators, blood metabolome, blood transcriptional patterns, microRNA, and blood genotoxicity markers were seen, according to an observational study to assess short-term molecular changes in swimmers. Nitrate is a precursor of nitric oxide, a free radical involved in the development of cancer and a contributor to intestinal inflammation. Notwithstanding this data, the biological plausibility of the relationships found with the risk of colorectal cancer is only partially supported by our knowledge of the mechanisms of action of nitrate and DBPs. We did a study to find immunological markers in blood associated with exposure to nitrate and trihalomethanes in drinking water and colorectal cancer risk. To calculate personal exposure indices, the concentration in drinking water was combined by the study individuals' residential municipality and year. Based on their homes since turning 18 and in the three years prior to the interview, except the past two years in both cases, study participants were given an average concentration of trihalomethanes(chloroform, bromodichloromethane, dibromochloromethane, and bromoform) and nitrate (i.e. year 3 prior the interview). Using individual data on the kind of water consumed (tap, bottled, private well), as well as the quantity (litres/day), exposure by ingestion was assessed. Those who reported drinking tap water received a residential level. Based on the literature, a zero value of THM and 6.1 mg/L of nitrate were assigned when respondents reported drinking bottled water (Espejo-Herrera et al., 2013,

Font-Ribera et al., 2010). Based on data from other study regions with ground water and THM measurements, values of 0.3 8 g/l, 0.3 8 g/l, 0.88 g/l, and 1.8 g/l, respectively, were assigned for chloroforom, bromodichloromethane, dibromochloromethane, and bromoform when individuals reported using well water. In the Leon region, where a well water test was done to detect nitrate, private well water is currently mostly consumed. Consumers of well water in this region were allocated levels from León well water samples, which ranged from 0.5 mg/L to 93 mg/L. Some places' well water was given missing values. Information about the exposure evaluation has been released in other places.

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