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THE IMPACT OF NUTRITIONAL BEHAVIOR ON CANCER RISK AND PROGRESSION

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ABSTRACT

Cancer remains one of the leading causes of mortality globally, following cardiovascular diseases. A growing body of evidence highlights the significant role that nutritional habits and dietary choices play in cancer development and progression. This review explores the association between food habits and cancer risk, with particular emphasis on the consumption of processed foods, genetically modified produce, pesticide-exposed products, and carcinogenic compounds present in various food items. Additionally, the study examines the synergistic effects of unhealthy lifestyle factors such as smoking, alcohol consumption, obesity, and sedentary behavior. Current research efforts are also discussed, focusing on the preventive role of dietary interventions and the promotion of healthier lifestyles as potential strategies to mitigate cancer risk. This comprehensive analysis underscores the critical importance of dietary habits in both the prevention and development of cancer.

INTRODUCTION

Globally, cancer constitutes the second leading cause of death after cardiovascular diseases. As a complex genetic disorder, cancer originates from mutations that cause uncontrolled proliferation of abnormal cells, eventually invading and compromising healthy tissues. These mutations may arise spontaneously or following exposure to carcinogens—substances that damage DNA or disrupt biological pathways crucial for cellular homeostasis.

Carcinogens are classified based on their origin: chemical, physical, or biological. Physical carcinogens include ultraviolet (UV) radiation, ionizing radiation, and X-rays; chemical carcinogens encompass compounds like cigarette smoke, alcohol, and certain hormones such as estrogens; biological carcinogens include viruses that integrate into host genomes. Endogenous factors, such as reactive oxygen species (ROS), can also induce oxidative stress, contributing to carcinogenesis. Food consumption is a significant exogenous factor influencing cancer risk. Cooking methods, consumption of genetically modified foods, and dietary exposure to various toxins can all contribute to DNA damage and subsequent cancer development. The International Agency for Research on Cancer (IARC) has evaluated over 1000 compounds, identifying numerous agents with varying degrees of carcinogenicity.

2. THE ROLE OF FOOD AND EATING HABITS IN CARCINOGENESIS

Nutritional factors are implicated in approximately 70% of all cancers and nearly 40% of cancer-related deaths (Key et al., 2004; Giovannucci et al., 2007) [1,7]. Numerous studies have investigated the carcinogenic potential of specific foods, food contaminants, and dietary behaviors. These factors not only directly affect cellular DNA but also indirectly modulate inflammation, hormonal balance, oxidative stress, and metabolic homeostasis, all of which contribute to cancer development and progression.

High-Calorie and High-Fat Diets

Excessive consumption of calorie-dense foods rich in refined sugars and saturated fats fosters obesity, which is a major risk factor for various cancers including breast, colorectal, pancreatic, and endometrial cancers (Donaldson, 2004; La Vecchia et al., 2015) [3,9]. Obesity promotes chronic low-grade inflammation by increasing the secretion of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and leptin, while reducing adiponectin levels. These hormonal imbalances lead to insulin resistance, increased levels of insulin-like growth factors (IGF), and altered estrogen metabolism, all of which create a pro-carcinogenic environment (McCullough & Giovannucci, 2004) [17].

Red and Processed Meats

Red meat is a well-established dietary risk factor due to its high heme iron content, which catalyzes the formation of reactive oxygen species and N-nitroso compounds (NOCs)—known genotoxic agents that cause DNA damage and promote tumorigenesis (Cross & Sinha, 2004; Bastide et al., 2011; Norat et al., 2005) [11,20,22]. The high-temperature cooking methods such as grilling, frying, and barbecuing further enhance the formation of heterocyclic amines (HCAs) and polycyclic aromatic

hydrocarbons (PAHs), both of which are potent mutagens (Turner & Lloyd, 2017) [23].

Dairy Products

While dairy products are rich in essential nutrients such as calcium and vitamin D, which may have protective effects against colorectal cancer, they also contain saturated fats and insulin-like growth factor-1 (IGF-1), which have been implicated in increased risks of prostate and breast cancers (Kushi et al., 2012) [13]. The dual nature of dairy underscores the complexity of dietary factors in cancer risk modulation.

Salted, Pickled, and Smoked Foods

Certain traditional preservation methods such as salting, pickling, and smoking introduce carcinogenic compounds like nitrosamines and polycyclic aromatic hydrocarbons (Goldenberg, 2002; Oliveira et al., 1995) [13,23]. High intake of these preserved foods has been particularly linked to gastrointestinal cancers, such as gastric and esophageal cancers, in populations where these practices are common (De Stefani et al., 2003) [22].

Alcohol Consumption

Alcohol is classified as a Group 1 carcinogen by the IARC and has been causally linked to multiple cancers including those of the oral cavity, pharynx, larynx, esophagus, liver, breast, and colorectum (Boffetta & Hashibe, 2006; Freedman et al., 2007) [4,18]. Ethanol metabolism produces acetaldehyde, a highly reactive and toxic compound that forms DNA adducts and interferes with DNA repair mechanisms, contributing to mutagenesis (Pöschl & Seitz, 2004) [25].

Mycotoxins and Foodborne Carcinogens

Aflatoxins, produced primarily by Aspergillus flavus and Aspergillus parasiticus in contaminated grains, nuts, and spices, are among the most potent naturally occurring carcinogens. Chronic exposure to aflatoxins is strongly associated with hepatocellular carcinoma, especially in synergy with hepatitis B virus infection (Peers & Linsell, 1973; Hayes et al., 1984) [22,13]. Other foodborne carcinogens include polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines, and dioxins, which enter the food chain through environmental contamination or food processing (Muir et al., 1992; Pitot, 2002) [23,24].

Lifestyle Factors Interacting with Diet

Lifestyle behaviors such as tobacco smoking, physical inactivity, and poor dietary habits act synergistically to amplify cancer risk (Parkin et al., 2011; Song & Giovannucci, 2016) [10,25]. Smoking combined with alcohol intake dramatically increases the risk for upper aerodigestive tract cancers, while sedentary behavior further contributes to metabolic dysfunction and systemic inflammation (Vineis et al., 2007) [29].

3. PROTECTIVE DIETARY COMPONENTS AND LIFESTYLE MODIFICATIONS

Conversely, certain dietary patterns and food components offer protective effects against cancer. The World Cancer Research Fund and the American Institute for Cancer Research recommend high-fiber diets rich in fruits, vegetables, and whole grains, while limiting red meat and high-fat foods.

Polyphenols—bioactive compounds found in fruits (e.g., dates, bananas, apples), green tea, and certain vegetables—exhibit potent antioxidant and anti-inflammatory properties, mitigating oxidative stress and inhibiting cancer-promoting pathways. Specific polyphenols have been shown to downregulate pro-inflammatory cytokines such as interleukin-6 (IL-6) and inhibit tumor growth through modulation of nuclear factor-kappa B (NF-κB) and cyclooxygenase pathways.

Dairy products may exhibit anti-carcinogenic effects due to the presence of vitamin D and lactoferrin, despite their potential risks. Overall, the quality and safety of food production, processing, and distribution remain essential in reducing carcinogen exposure and promoting cancer prevention.

II.LITERATURE REVIEW

The relationship between nutritional behavior and cancer development has been extensively studied over the past decades. Numerous epidemiological, clinical, and experimental studies have established that dietary patterns substantially influence cancer risk and progression through various mechanisms involving oxidative stress, inflammation, hormonal modulation, and genotoxicity.Several large-scale studies and expert reviews have demonstrated that unhealthy diets rich in processed foods, red meats, alcohol, saturated fats, and food contaminants contribute significantly to the global cancer burden (Key et al., 2004; Wiseman, 2008; Boffetta & Hashibe, 2006; Cross & Sinha, 2004) [1, 4, 6, 11]. Red and processed meats, in particular, have been associated with colorectal cancer due to the formation of heme iron-derived N-nitroso compounds and heterocyclic amines during high-temperature cooking (Chan et al., 2011; Bastide et al., 2011; Turner & Lloyd, 2017; Schwingshackl et al., 2017) [12, 20, 23, 24]. Additionally, high intake of saturated fats and calorie-dense diets contributes to obesity, which is a well-established risk factor for hormone-sensitive cancers such as breast and endometrial cancers (Donaldson, 2004; La Vecchia et al., 2015) [3, 9].

Alcohol consumption has also been consistently linked to elevated cancer risk, particularly for cancers of the head, neck, liver, breast, and esophagus (Boffetta & Hashibe, 2006; Freedman et al., 2007) [4, 18]. Mechanistically, alcohol's carcinogenic potential is largely mediated through its metabolite acetaldehyde and its capacity to increase oxidative stress (Liu, 2004) [5].

On the other hand, diets abundant in fruits, vegetables, dietary fibers, and bioactive phytochemicals have shown protective effects against carcinogenesis by modulating oxidative damage, inflammation, and hormonal pathways (Giovannucci et al., 2007; Farvid et al., 2014; Bradbury et al., 2014) [7, 16, 27]. Polyphenols, carotenoids, flavonoids, and other antioxidant compounds found in plant-based foods help to neutralize reactive oxygen species, thereby reducing DNA damage (Liu, 2004; Zhang et al., 1999) [5, 8].

The World Cancer Research Fund and American Institute for Cancer Research have extensively evaluated the modifiable factors contributing to cancer, emphasizing the importance of maintaining a healthy weight, engaging in regular physical activity, and consuming a diet rich in plant-based foods (Wiseman, 2008; Kushi et al., 2012) [6, 13]. Evidence from case-control and cohort studies in diverse populations, including Asian, Mediterranean, and Western cohorts, underscores the consistency of these findings across different ethnic and geographic settings (Zhang & Holman, 2011; Seow et al., 2002; Norat et al., 2005; Song & Giovannucci, 2016) [2, 21, 22, 25].

Moreover, emerging studies have explored the molecular mechanisms through which dietary factors influence carcinogenesis, including modulation of the Nrf2 pathway, NF-κB activation, and inflammatory cytokine regulation (Dashwood, 2007; McCullough & Giovannucci, 2004; Ferguson, 2010) [14, 17, 15]. Nutrient-based dietary patterns and nutrient interactions further suggest that cancer prevention may not rely solely on individual nutrients but on complex dietary patterns as a whole (Edefonti et al., 2010) [30].

Lifestyle factors, such as smoking and sedentary behavior, synergistically interact with dietary risk factors, further increasing the risk for multiple cancer types (Parkin et al., 2011; Vineis et al., 2007) [10, 29]. Tobacco, in particular, remains one of the most significant lifestyle-related contributors to cancer risk worldwide.

CONSLUSION

Dietary habits play a critical role in both the development and prevention of cancer. Many modern dietary patterns expose individuals to carcinogenic compounds, emphasizing the urgent need for public health interventions that promote safer food practices and healthier lifestyles. Continued research is essential to better understand these relationships and guide policy-making in cancer prevention strategies. Educational programs focusing on dietary improvements and food safety can significantly contribute to reducing global cancer burdens and improving overall quality of life.

Nutritional behavior plays a pivotal role in both the initiation and progression of cancer. Dietary patterns characterized by high consumption of processed foods, red and processed meats, alcohol, saturated fats, and carcinogenic contaminants have been consistently associated with elevated cancer risk. Conversely, diets rich in fruits, vegetables, fiber, poly phenols, and essential micronutrients exhibit protective effects through anti-inflammatory, antioxidant, and anti-proliferative mechanisms. In addition to specific food components, overall

lifestyle factors—including physical activity, body weight management, and avoidance of tobacco and alcohol—further modulate individual susceptibility to cancer.

This review underscores the importance of adopting balanced dietary habits and implementing food safety measures as critical strategies for cancer prevention and control. Public health policies aimed at increasing awareness, promoting healthy eating, and regulating exposure to dietary carcinogens are essential to reduce the global cancer burden. Future research should continue to explore the complex interplay between nutrition, genetic predisposition, and environmental exposures to better inform personalized cancer prevention and management strategies.

REFERENCES

- Key, T. J., et al. (2004). Diet, nutrition and the prevention of cancer. *Public Health Nutrition*, 7(1A), 187–200.
- Zhang, Y., & Holman, C. D. (2011). Dietary risk factors for breast cancer in Chinese women. *Asian Pacific Journal of Cancer Prevention*, 12(10), 2467-2472.
- Donaldson, M. S. (2004). Nutrition and cancer: A review of the evidence for an anti-cancer diet. *Nutrition Journal*, 3(1), 19.
- Boffetta, P., & Hashibe, M. (2006). Alcohol and cancer. *The Lancet Oncology*, 7(2), 149-156.
- Liu, R. H. (2004). Potential synergy of phytochemicals in cancer prevention: Mechanism of action. *The Journal of Nutrition*, 134(12), 3479S-3485S.
- Wiseman, M. (2008). The second World Cancer Research Fund/American Institute for Cancer Research expert report. *Proceedings of the Nutrition Society*, 67(3), 253-256.
- Giovannucci, E., et al. (2007). Nutrition, physical activity, and cancer: A review of the evidence. *Journal of the National Cancer Institute*, 99(14), 1171-1193.
- Zhang, S., Hunter, D. J., et al. (1999). Dietary carotenoids and risk of breast cancer. *American Journal of Clinical Nutrition*, 70(3), 509-516.
- La Vecchia, C., et al. (2015). Diet and cancer risk in Mediterranean countries: results from a series of case-control studies. *International Journal of Cancer*, 137(4), 935-945.
- Parkin, D. M., Boyd, L., & Walker, L. C. (2011). 16. The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010. *British Journal of Cancer*, 105, S77–S81.
- Cross, A. J., & Sinha, R. (2004). Meat-related mutagens/carcinogens in the etiology of colorectal cancer. *Environmental and Molecular Mutagenesis*, 44(1), 44-55.
- Chan, D. S. M., et al. (2011). Red and processed meat and colorectal cancer incidence: meta-analysis of prospective studies. *PloS One*, 6(6), e20456.
- Kushi, L. H., Doyle, C., et al. (2012). American Cancer Society guidelines on nutrition and physical activity for cancer prevention. *CA: A Cancer Journal for Clinicians*, 62(1), 30-67.
- Dashwood, R. H. (2007). Nrf2 as a target for cancer chemoprevention. *Current Cancer Drug Targets*, 7(5), 393-409.
- Ferguson, L. R. (2010). Meat and cancer. *Meat Science*, 84(2), 308-313.
- Farvid, M. S., et al. (2014). Fruit and vegetable consumption and risk of breast cancer. *Breast Cancer Research and Treatment*, 144(2), 465-473.
- McCullough, M. L., & Giovannucci, E. L. (2004). Diet and cancer prevention. *Oncology (Williston Park)*, 18(11), 1173-1182.
- Freedman, N. D., et al. (2007). Alcohol and head and neck cancer risk in a prospective study. *British Journal of Cancer*, 96(9), 1469-1474.
- Chan, A. T., & Giovannucci, E. L. (2010). Primary prevention of colorectal cancer. *Gastroenterology*, 138(6), 2029-2043.
- Bastide, N. M., et al. (2011). Heme iron from meat and risk of colorectal adenomas. *American Journal of Clinical Nutrition*, 94(2), 467-474.
- Seow, A., et al. (2002). Diet, reproductive factors and lung cancer risk among Chinese women in Singapore. *Cancer Causes & Control*, 13(1), 35-43.
- Norat, T., et al. (2005). Meat, fish, and colorectal cancer risk: the European Prospective Investigation into cancer and nutrition. *Journal of the National Cancer Institute*, 97(12), 906-916.
- Turner, N. D., & Lloyd, S. K. (2017). Association between red meat consumption and colon cancer: a systematic review of experimental results. *Experimental Biology and Medicine*, 242(8), 813-839.
- Schwingshackl, L., et al. (2017). Dietary intake and risk of colorectal cancer: a systematic review and meta-analysis. *Nutrition Journal*, 16(1), 26.
- Song, M., & Giovannucci, E. L. (2016). Preventable incidence and mortality of carcinoma associated with lifestyle factors. *JAMA Oncology*, 2(9), 1154-1161.
- Lin, J., et al. (2005). Intakes of fruits, vegetables, and related nutrients and the risk of colorectal cancer. *Journal of the National Cancer Institute*, 97(22), 1630-1637.
- Bradbury, K. E., et al. (2014). Fruit, vegetable, and fiber intake in relation to cancer risk. *International Journal of Epidemiology*, 43(6), 1815-1825.
- Boyle, P., & Levin, B. (Eds.). (2008). *World Cancer Report 2008*. IARC.
- Vineis, P., et al. (2007). Tobacco and cancer: recent epidemiological evidence. *Journal of the National Cancer Institute*, 99(7), 472-490.
- Edefonti, V., et al. (2010). Nutrient-based dietary patterns and cancer risk: a review. *Nutrition Reviews*, 68(9), 519-532.