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The Role Of Dietary Choices In Cancer Risk: A Comprehensive Analysis Of The Influence Of Red Meat, Alcohol, Dietary Fibres And Probiotic Consumption

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I. Introduction:

Cancer remains a major global health challenge, affecting millions of individuals worldwide. Globally, colorectal cancer (CRC) ranks as the third most frequently diagnosed cancer among men, accounting for approximately 746,000 new cases annually, and the second most prevalent among women, with approximately 614,000 new cases reported each year. It is also the fourth highest cause of cancer death. In recent years, there has been a growing recognition of the significant role that lifestyle factors play in influencing cancer risk. Dietary factors are likely to exert an impact on the process of colorectal carcinogenesis through a complex interplay of multiple mechanisms. This exploration delves into the impact of specific lifestyle choices, dietary habits and alcohol consumption, on cancer risk. By conducting an extensive review of scientific literature and epidemiological studies, the paper aims to provide a comprehensive analysis of how these lifestyle factors can modulate cancer risk and contribute to the development of effective public health strategies for cancer prevention.

In the year 1981, Doll and Peto estimated that up to 35% of deaths related to cancer, and as much as 90% of deaths specifically caused by stomach and large bowel cancers, could potentially be linked to factors related to one's dietary choices. In this investigation, the link between different factors of one's diet and alcohol consumption can raise the likelihood of developing cancer.

The complex link between dietary elements and cancer risk was explored in this literature review, with a particular emphasis on red meat intake, alcohol consumption, and dietary fibres. Despite being a source of important nutrients, red meat has been associated with a higher risk of colorectal cancer (CRC) because it contains hazardous substances, such as heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons (PAHs), and nitroso compounds (NOCs). According to recent meta-analyses, each additional 100 g/day of red meat consumption is related to a 20% increased risk of CRC. Additionally, eating red meat increases the production of hydrogen sulphide (H2S) and amino acids that contain sulphur, both of which aid in cancer development. Additionally, red meat consumption-related oxidative stress and inflammation can induce DNA damage and contribute to a number of chronic diseases.

Alcohol intake is a substantial risk factor for a number of malignancies, including oropharyngeal, esophageal, liver (hepatocellular carcinoma), breast, and colorectal cancers. By attaching to DNA and proteins, alcohol breakdown products, particularly acetaldehyde, have mutagenic and carcinogenic effects. Despite evidence pointing to a possible decline in cancer risk during protracted abstinence from alcohol, thorough information on this subject is still lacking.

However, a high-fibre diet seems to prevent cancer. Short-chain fatty acids (SCFAs), which are produced as a result of dietary fibre intake, inhibit histone deacetylase, control gene expression, and slow carcinogenesis. Additionally, SCFAs work with G protein-coupled receptors to reduce gut inflammation and to support immunological homeostasis. According to epidemiological studies, a high-fibre diet may help prevent gastrointestinal, breast, ovarian, endometrial, and other cancers.

II. Factors Affecting Prognosis

Red Meat Consumption

Red meat is an essential component in the diet of the majority of the population. Red meat contains a lot of essential nutrients like protein, amino acids, fats, vitamins and minerals such as iron, magnesium and zinc. In addition to that, meat is also rich in chemicals like heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons (PAHs), nitrate and N-nitroso compounds (NOCs), which scientists have discovered to cause an increased risk to multiple types of cancer. Long-term meat consumption is a prominent factor for the increased risk of cancer in the distal portion of the large intestine and is known as a carcinogen for colorectal cancer (CRC). The World Cancer Research Fund, through their research, has found convincing evidence that the consumption of Red Meat

can increase the risk of CRC. According to a recent meta-analysis, there appears to be a roughly 20% elevated risk of colorectal cancer (CRC) associated with each additional intake of 100 g/day of red meat and 50 g/day of processed meat. The consumption of red meat increases the concentration of sulphur-containing amino acids in the blood. These propagate the growth of H_2S. New functions of H_2S in the progression of cancer have come to light in recent studies, shedding fresh insight into the disease's underlying mechanisms. An increase in H_2S production has also been linked with increased cell proliferation. The overconsumption of Red Meat and its processed counterparts can also lead to surplus saturated fat, which in turn can cause the excess production of bile acid in the stomach. The buildup of the same can cause one to become overweight or obese and would lead to comorbidities as well as cancer risk.

Additionally, red meat consumption induces mutations which cause degradation of DNA and N-nitroso compounds (NOCs) to be introduced in one's system, which are potent carcinogens in the gastrointestinal tract. Processed meats also introduce exogenous NOCs due to the addition of nitrates and nitrites during the preservation process. NOCs also induce the release of Heme-Iron which adds oxidative stress to the body. This oxidative stress can lead to DNA damage and inflammation. Persistent oxidative stress can potentially give rise to prolonged inflammation, which, in a cascading manner, might play a pivotal role in facilitating numerous chronic conditions. Additionally, the production of heme-iron may lead to an increase in mucin-degrading bacteria (*Akkermansia muciniphila*) causing damage to the function of the gut barrier. This leads to diseases including but not limited to cancer, diabetes, cardiovascular issues, neurological disorders, and pulmonary diseases.

The inflammatory cells that are caused by oxidative stress generate reactive oxygen species (ROS) and reactive nitrogen intermediates (RNI). ROS and RNI potentially induce mutations in nearby epithelial cells. Additionally, the cytokines released by inflammatory cells can amplify the levels of intracellular ROS and RNI within pre-malignant cells. Inflammation also has the potential to bring about epigenetic modifications that promote the initiation of tumours. The inflammation associated with tumours perpetuates a cycle wherein there is heightened production of ROS, RNI, and cytokines.

Alcohol Consumption

Alcohol Consumption is a causal link factor to the development of cancers affecting the oropharynx and larynx, oesophagus, liver (hepatocellular carcinoma), breast, and colorectal cancer. In the year 2012, approximately 5.5% of newly diagnosed cancer cases and 5.8% of global cancer-related deaths were projected to be linked to alcohol consumption. In the United States, calculations suggest that about 3.5% of all cancer-related deaths can be attributed to alcohol consumption. A meta-analysis of 27 cohorts and 34 case-control studies in regions such as the USA, Europe, Asia, and Australia showed strong evidence for a positive association between alcohol drinking of >1 drink/day and CRC risk.

Relative Risk (95% CI)

Type of Cancer	Nondrinker	Light Drinker	Moderate Drinker	Heavy Drinker
Oral cavity and pharynx	1.0 (referent)	1.13 (1.0 to 1.26)	1.83 (1.62 to 2.07)	5.13 (4.31 to 6.10)
Esophageal squamous cell carcinoma	1.0 (referent)	1.26 (1.06 to 1.50)	2.23 (1.87 to 2.65)	4.95 (3.86 to 6.34)
Larynx	1.0 (referent)	0.87 (0.68 to 1.11)	1.44 (1.25 to 1.66)	2.65 (2.19 to 3.19)
Liver	1.0 (referent)	1.00 (0.85 to 1.18)	1.08 (0.97 to 1.20)	2.07 (1.66 to 2.58)
Female breast	1.0 (referent)	1.04 (1.01 to 1.07)	1.23 (1.19 to 1.28)	1.61 (1.33 to 1.94)
Colorectum	1.0 (referent)	0.99 (0.95 to 1.04)	1.17 (1.11 to 1.24)	1.44 (1.25 to 1.65)

Adapted from the results of Bagnardi et al (2015).

Post consumption of alcohol, the alcohol dehydrogenase and aldehyde dehydrogenase catalysed reaction are the start of carcinogenesis. The initial breakdown of ethanol by its oxidation into acetaldehyde and then acetate. Although ethanol is not carcinogenic, its product acetaldehyde exhibits mutagenic and carcinogenic properties by binding to DNA and protein.

The findings from these studies indicate that the potential risk of cancer could potentially decrease to the level observed in individuals who have never consumed alcohol, following prolonged abstinence from alcohol consumption lasting 20 years or more. Unfortunately, the lack of comprehensive data concerning the impact of discontinuing alcohol intake causes the impact to be questioned. This subject, which holds significance, requires a more comprehensive examination. Another study brings light to individuals who have chosen to abstain from

alcohol consumption and how their cancer risk might differ from current drinkers. To provide an example, similar to abstinence from smoking, a heightened near-term cancer risk after abstinence from alcohol consumption might result from the emergence of symptoms related to cancer.

Dietary fibres and probiotics

A high-fibre diet may help one eat less calories in general and retain a healthy weight, both of which are essential for minimising risk for developing cancer. Additionally, research suggests that eating a diet rich in fibre may help prevent gastrointestinal, breast, ovarian, endometrial, and other cancers. The epidemiologic evidence showing an inverse relationship between a high-fibre diet and risk for the malignancies described above is the main topic of this review.

Through the consumption of dietary fibres, fatty acids having fewer than six carbon atoms, referred to as short-chain fatty acids, are produced. They are the primary source of energy for the cells lining your colon and are created when the good gut bacteria ferment fibre in your colon. The production of short chain fatty acids causes the inhibition of the histone deacetylase. This propagates the epigenetic regulation of gene expression which limits tumour formation. Additionally, short-chain fatty acids interact with G protein-coupled receptors. The metabolite-sensing G protein-coupled receptors GPR41, GPR43, and GPR109A, which are expressed in the gut epithelium and in immune cells, interact with SCFAs. These interactions trigger processes that are essential for preserving homeostasis in the stomach and other organs. This interaction limits inflammation of the gut and reduces development of CRC. SCFAs are also considered bacteria signalling and they modify the metabolism and epigenetic state of host T cells in order to cause an effect. They play a crucial biological role in encouraging (intestinal) regulatory T cell development and in the generation of the anti-inflammatory cytokine interleukin-10. This boosts the intestinal immune homeostasis and can decrease CRC.

An unbalanced gut microbiome can cause inflammatory immune system reactions and contribute to the development of diseases, including cancer. Certain probiotic strains have been used as adjuvant therapies during chemotherapy for cancer by modifying the intestinal microbiota and immune response.

The human gut microbiome benefits from the use of probiotics. Their primary benefit is their impact on the growth of the microbiota that inhabits the organism, which ensures a good balance between the bacteria required for an organism to operate normally and pathogens. The preservation and restoration of intestinal homeostasis is made possible by the beneficial effects of probiotics. Live bacteria, known as probiotics, can help host health when administered at sufficient doses. In the human diet, silage (such as cabbage and cucumbers) and fermented milk products (such as yoghurt and kefir) are the main sources of probiotics.

III. Conclusion

With millions of new cases each year, cancer, especially colorectal cancer (CRC), presents a huge health challenge worldwide. Cancer risk is greatly influenced by lifestyle variables including dietary choices and alcohol consumption. Consumption of red meat, which is high in carcinogens such as HCAs, PAHs, and NOCs, is associated with a 20% higher risk of CRC per 100 g/day. It also increases the levels of amino acids, including sulphur and hydrogen sulphide, which promote cancer growth. Drinking is a significant risk factor for many malignancies owing to the carcinogenic acetaldehyde breakdown product of alcohol. Long-term abstinence may lower the risk of cancer development; however, further in-depth research is required. A high-fibre diet, on the other hand, suppresses histone deacetylase, limiting carcinogenesis and reducing inflammation by creating short-chain fatty acids. SCFAs support gut health and reduce CRC by interacting with GPRs. Probiotics aid in preserving a healthy gut microbiome by reducing inflammatory responses that contribute to cancer growth. In conclusion, nutrition, alcohol use, and dietary fibre have a significant impact on cancer risk, providing important information for public health initiatives and cancer prevention.

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