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Review article

Risk factors and lifestyle factors in the development and prevention of cancer: A literature review

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Abstract: Cancer represents a major global health challenge, with steadily increasing incidence rates worldwide. A hallmark of cancer development is metabolic reprogramming, whereby cancer cells alter their metabolic behavior to meet the demands of energy production, biosynthesis, and redox homeostasis, distinguishing them from normal cells. Numerous environmental, behavioral, and biological factors contribute to cancer risk, including alcohol consumption, tobacco use, frequent intake of fast foods, exposure to environmental pollutants, viral infections such as human immunodeficiency virus (HIV), psychological stress, and dysregulation of key metabolic pathways involving glucose, amino acids, and fatty acids. Cancer progression involves the transformation of normal cells into highly malignant and invasive tumors, often accompanied by genomic instability. Emerging evidence suggests that modifiable lifestyle factors play a crucial role in both the development and prevention of cancer. Adoption of healthy dietary patterns, calorie restriction, regular physical activity, cessation of smoking and alcohol use, and avoidance of obesity-promoting behaviors such as excessive consumption of fatty foods and hot beverages can help regulate metabolic alterations and significantly reduce cancer risk. This review highlights the interplay between risk factors, metabolic changes, and lifestyle interventions in cancer development and prevention.

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Introduction

Cancer, a hereditary disease, occurs when tumor cells deviate from their progenitor cells [1]. These genetic changes affect cellular growth genes. Sequential mutations in cellular genes activate oncogenes and deactivate tumor suppressor genes, causing malignant transformation. Modifiable risk factors for malignancies include diet, tobacco, weight, alcohol, and smoking, as well as physical inactivity, drug use, sexual and reproductive health, exposure to air pollution, and use of infected needles are the key environmental and behavioural risk factors for cancer mortality worldwide [2]. Research on the benefits of integrating chemo-preventive foods has exploded in recent decades. Numerous studies have shown that chemo-preventive drugs have lasting anti-carcinogenic capabilities that can disrupt multiple carcinogenesis processes [3]. People who abuse or are dependent on alcohol frequently smoke cigarettes; in fact, up to 80% of people who are addicted to alcohol are also cigarette smokers. The combined exposure to alcohol and tobacco smoke has adverse health effects, including increased risk for some diseases like head and neck cancer.

Smoking often cancels out the good health effects of moderate alcohol use. Molecular epidemiology techniques are shedding light on the significance of effects on oxidant/antioxidant pathways and on metabolic pathways involving the liver, which are altered by combined smoking and alcohol exposure [4]. While smoking, which may account for 20% to 30% of all incidence malignancies, is undoubtedly the most significant lifestyle-related risk factor overall, it varies

widely depending on the type of cancer and its subtypes [5]. Surprisingly, it has been suggested that quitting smoking, consuming less alcohol, maintaining a healthy weight through exercise, eating a plant-based diet, and breastfeeding could prevent 50% of all cancers in industrially developed and wealthy countries [6]. High salt intake and consumption of salt-preserved foods may raise the risk of stomach cancer; overall salt and salt-preserved food consumption should be modest.

Drinks and foods shouldn't be ingested when scorching hot because they may raise the risk of mouth, pharynx, and oesophagus malignancies. 76% of lung cancer fatalities are caused by smoking, eating few fruits and vegetables, urban air pollution, and indoor smoke from solid fuel use in the home [7]. The American Chemical Society (ACS) Guidelines concentrate on suggestions for individual decisions about diet and exercise regimens. Still, these decisions occur in a social framework that supports or hinders healthy behaviours [8]. According to the 2010 Dietary Guidelines for Americans and the 2008 Physical Activity Guidelines for Americans, the ACS Guidelines are consistent with recommendations from the American Heart Association and the American Diabetes Association for the prevention of coronary heart disease and diabetes as well as for the promotion of general health [9]. Cancer prevention requires smoking cessation, increased ingestion of fruits and vegetables, moderate use of alcohol, caloric restriction, exercise, avoidance of direct exposure to sunlight, minimal meat consumption, use of whole grains, use of vaccinations, and regular check-ups [10]. An effective, yet straightforward, method for converting epidemiologic findings into primary cancer prevention is the use of combinations of modifiable lifestyle factors in Colorectal Cancer (CRC) risk assessment [11].

Little concrete evidence addresses how weight loss affects cancer risk because few obese persons successfully lose weight over the long term [12]. Therefore, although lifestyle factors like diet, alcohol, physical inactivity, and obesity are responsible for 50% of colorectal cancer cases identified in the U.K. in 2010, it has been predicted that only around half of these instances are prevented in a realistic (20-year) timeframe [13]. There is a lack of information on the potential increases in cancer risk brought on by widowhood and divorce, two rising demographic trends [14]. The cancer risk for divorced people was 0.92 to 0.94, with increased risks for sites associated with drinking, tobacco use, and the human papillomavirus and decreased risks in most other areas [15]. According to the research, practically every type of cancer is affected by lifestyle changes and a spouse's death. Due to the size of the impacts, epidemiological studies that do not consider marital status may be biased [16]. Although its significance in the absence of hepatitis virus infections is unclear, aflatoxin exposure in foods should be minimized as it promotes liver cancer [17]. Chinese-style salted fish should only be consumed in moderation as it raises the risk of nasopharyngeal cancer, especially during childhood [18]. Fruits and vegetables likely lower the incidence of oesophageal, oral, and other types of cancer. Diets should contain at least 400 g/d of total fruits and vegetables to support the stomach and colorectum. Red meat and preserved meat consumption should be limited since they may raise the risk of colorectal cancer. Regular physical exercise should be practiced because it is the primary determinant of energy expenditure and lowers the risk of breast and colorectal cancer [19]. Globally, the incidence of cancer is rising. It has been suggested that alteration of the metabolic pathway distinguishes cancer from other types of cells regarding how nutrients are used. Changes in the metabolism of nutrients like glucose, amino acids, and fatty acids are linked to an increased risk of developing cancer. Calorie restriction and regular exercise improve lifestyle practices that can lower cancer risk [20]. The colonic epithelium's age-associated DNA methylation alteration is moderated by lifestyle factors, such as aspirin use, and affects the development of cancer methylomes [21]. Almost 170 observational epidemiologic studies have investigated the relationship between physical activity and cancer risk at various cancer locations. According to the information that is now available, specific public health organizations have published physical activity recommendations for cancer prevention, often suggesting at least 30 minutes of vigorous to moderate physical activity five days a week. Although most studies have concentrated on the effectiveness of exercise in preventing cancer, there is mounting evidence that exercise also impacts other elements of the cancer experience, such as cancer detection, coping, rehabilitation, and survival after diagnosis [22].

Impact of lifestyle on cancer

The genes transferred from parents to offspring have very little effect on offspring's life [23]. Human biology is complicated as it deals with many independent health factors. Genes can transfer information from one generation to another generation, and if mutation occurs, it may cause diseases. On the other hand, some external factors can cause serious consequences. For example, only 20% of breast cancer is found in twins due to genetic mutation, and different environmental factors cause mutation. In comparison, 90% of cancer

can be caused by environmental factors and lifestyle [24]. Lifestyle effects like smoking, Diet, use of alcohol, different types of infections, radiation and pollutants.

The World Health Organization (WHO) evaluation focuses on lifestyle exposures contributing to cancer, including addictive substances, Diet, physical inactivity, sexual and reproductive health, environmental risks, and exposure to contaminated needles/injections in healthcare institutes [25]. The joint effect of these preventable risk factors is about 35%, or 2.43 million cancer deaths worldwide in 2001 [26]. Most cancer deaths are attributed to lifestyle factors in low-and middle-income countries, with smoking causing 21% of cancer deaths worldwide [27]. Low fruit and vegetable consumption causes 5% of all cancer deaths worldwide, while alcohol use causes 5% [28]. Lifestyle factors contribute to 41% of cancer deaths in men, with lung and cervix uteri cancers being the most preventable [29].

Harmful lifestyle leading to cancer development

Cancer is one of several non-communicable (NCDs) illnesses that will become more of a burden in low and middle-income countries (LMICs) as a result of the current epidemiological change [30]. Cancer was responsible for 29% of all fatalities caused by NCDs worldwide in 2001 [31]. The declining risks of infectious illnesses in an older population and improved therapeutic care of cardiovascular disorders are significant factors in the rising burden of cancer. Lifestyle changes can also cause shifts in cancer incidence rates, frequently in an upward direction. The global cancer burden is expected to reach 21.4 million new cases and 13.2 million deaths by 2030 [32]. Cancer rates are expected to rise rapidly in LMICs, especially in China, India, the rest of Asia, Africa, Latin America and the Caribbean [30].

Effect of cancer on widowhood and divorce

According to the available data, divorce may increase the chance of developing some forms of cancer, including pancreatic and prostate cancer [33]. It has also been theorized that people who live alone are at an increased risk of developing malignancies due to drug abuse and sexual activity. Divorcees may be less likely to get malignancies like colon and melanoma since they have less disposable income than those who have never been married. The Swedish Family Cancer Database was used for the analysis in this study. The Family-Cancer Database, which includes data from the 1960, 1970, 1980, and 1990 national censuses, was used for the analysis [34]. In the 1960 and 1970 censuses, the researchers looked only at those with the same civil status [35]. Cancers of the upper digestive tract, leukaemia, rectal cancer, and squamous cell carcinoma of the skin were among those the research sought to investigate. Cancer diagnoses, mortality rates, international migration, and the final study date were all recorded during the follow-up period that began in 1971 and concluded in 1998 [36]. The ratio of observed instances to those predicted was estimated using standard incidence rates (SIR). Further adjustments were made for parity and age at first birth for female malignancies. Cancer locations with at least 50 incidences were reported for the single and divorced population [37].

According to the research, divorce and widowhood are rising in many different cultures. Widows and divorced women made up 13–15% of the population of the same gender in 1990, while widowers made up 4.6% of the population [38]. Divorce rates also shifted over time, with the number of newly divorced people tripling between 1970 and 1980 [39]. Long-term singleness due to death or divorce was correlated with an increased cancer risk in this study. The results demonstrated that divorce's impact on cancer risks was more pronounced for divorcees than for widows and widowers. Divorce was associated with an increased risk of the upper digestive tract, pancreatic, lung, oesophageal, and cervical cancers among the study's participants [40]. Factors such as cigarette use, alcohol intake, and human papillomavirus infection were linked to these elevated danger levels. However, those who had been divorced were less likely to get cancers of the colon, kidney, skin, neurological system, or endocrine glands. Affluence was linked to a lower risk of these conditions. According to the study, cancer rates among widowed and divorced are comparable to those of other socially disadvantaged populations [41].

Devastating Impact of tobacco use

Tobacco use is a significant risk factor for cancer development [42]. It contains thousands of compounds, some carcinogens that can cause DNA mutations, epigenetic changes, and inflammation. An estimated 90 percent of lung cancer cases in nations with a high Gross Domestic Product (GDP) are attributed to smoking [43]. Additionally, it is associated with cancers of the pharynx, larynx, oesophagus, pancreas, kidney, and urinary tract. The greater the duration and intensity of smoking, the greater the risk of developing cancer [44]. The risk can be reduced by quitting smoking, but it may not decline to the level of never smokers. Passive smoking, or exposure to secondary smoke, has also been linked to an increased risk of cancer, albeit a lesser risk.

Continuing to smoke after a cancer diagnosis can diminish the efficacy of treatment, increase the risk of second primary malignancies, and negatively influence survival and quality of life [45].

Examining the link between alcohol consumption and smoking

Based on the information, heavy alcohol consumption and cigarette smoking are strongly correlated, with 80 percent of alcoholics also being smokers [46]. Smokers are 10 times more likely to develop alcoholism, which can worsen nicotine dependence [47]. Both smoking and drinking increase the risk of developing certain types of cancer, particularly cervical and head cancers. Additionally, alcohol and smoking exposure can have significant impacts on non-smokers. Specific genotypes, such as arylamine N-acetyltransferases (NAT1 and NAT2), have a role in laryngeal cancer susceptibility [48]. Mutations in the p53 gene are more frequent in the tumours of individuals who consume alcohol and smoke cigarettes [49]. The influence of combined alcohol and tobacco smoke exposure on oxidant injury and antioxidant defence systems is examined, including the role of glutathione-S-transferases M1 (GSTM1) null genotype in oesophageal cancer [50].

Tobacco

In 1964, smoking was first identified as a primary source of lung cancer in the USA. Then, the use of tobacco decreased. Tobacco may cause 14 types of cancer, like larynx, lung, oropharynx, etc. The current number of smokers worldwide is about 76% [51].

Alcohol

In 1910, the 1st case studied. In this case, the linkage between Alcohol consumption and a higher chance of oesophageal cancer was studied [52]. Long-term use of alcohol can cause UDT cancers in the oesophagus, larynx, oral cavity, etc. Alcohol can also cause breast cancer and pancreas cancer [53].

Alcohol consumption is strongly linked to the development of certain types of cancer, including cancers of the mouth, pharynx, larynx, oesophagus, liver, stomach, gallbladder, pancreas, colorectal, breast, and lung. Heavy drinkers have a higher risk of these cancers compared to non-drinkers. Approximately 4% of all cancers can be attributed to daily alcohol consumption, with higher percentages for certain types of cancer [54]. Breast cancer is particularly affected, with 60% of all alcohol-related cancers in women occurring in the breast. There is also a strong interaction between alcohol consumption and smoking, increasing the risk of cancer in the upper digestive tract [55].

Obesity and cancer

American Cancer Society (ACS) revealed that obesity can increase cancer risk and cancer of the breast, kidneys, liver cancer, etc. [56] Due to being overweight 14% chances of cancer in men while 20% chances in women [57]. A significant reason for overweight is fast food and junk food. Based on the studies, there is a large body of epidemiological evidence linking body fat content, as measured by body mass index (BMI), to cancer susceptibility. An elevated BMI has been shown to be associated with an increased risk of developing cancer [58]. These include gastrointestinal adenocarcinoma, gallbladder, endometrial, kidney, thyroid, colon, liver (in male inmates), leukaemia, myeloma, non-Hodgkin lymphoma, breast cancer after menopause, pancreatic cancer, melanoma, and rectal cancer. BMI inversely affects premenopausal and lung cancer risk. This relationship is mostly seen in smokers. BMI is inversely correlated with restricted carcinomas and directly correlated with advanced prostate cancer [59]. Overweight at diagnosis is associated with poorer cancer outcomes, particularly in breast cancer patients. Uterine corpus, postmenopausal breast, and colon cancer had the highest BMI associated cancer rates. Obesity-related oestrogen, insulin, and inflammation may increase cancer risk [60].

Obesity is strongly linked to several types of cancer, including colon, female breast, endometrium, kidney, and oesophagus cancers, according to the International Agency for Research on Cancer [61]. Overeating is significantly avoidable cause of cancer in non-smokers. Few obese individuals achieve long-term weight loss, and there is limited evidence on how weight loss affects cancer risk. Obesity is linked to a significant number of cancer deaths in the U.S., with one in seven deaths in men and one in five deaths in women being attributed to overweight and obesity. BMI is a metric used to classify weight and height in the US adult population [62]. The assumption is that true adiposity is not connected to height. Standards for healthy weight, overweight, and obesity have changed over time based on our understanding of weight's impact on health [63]. Self-reported data on weight and height is closely related to actual measurements, but estimates of overweight and obesity tend to be lower when based on self-reports compared to actual measurements. BMI may not accurately reflect body fat in older adults due to changes in fat distribution. As people age, they tend to accumulate more fat around their waist, which can affect their waist-to-hip ratio. This means that even if

their BMI remains the same, their body composition may have changed [64].

Obesity-causing mutations in humans are rare, first discovered through single-gene changes in rats. Recent advancements in genome knowledge and high-throughput genotyping have facilitated investigating the connection between common polymorphisms and obesity. Scientists have identified genes contributing to obesity predisposition using beta-adrenergic receptors, PPARg, LEP, LEPR, uncoupling protein genes, and TNFa [65].

- Insulin-like growth factors (IGFs) are mitogens (peptide which induce cells to begin cell division) that enhance cell proliferation and prevent apoptosis.
- Obesity is linked to endometrial, renal, oesophageal, and other malignancies.
- Overweight and obesity are the leading causes of U.S. cancer fatalities, measured by population attributable fraction (PAF). Most studies demonstrate poor long-term weight maintenance [66].

Interventions must understand the behaviours and variables that facilitate weight reduction. Since 1994, the National Weight Control Registry (NWCR) has enrolled 3000 US adult men and women who have maintained a 13.6kg weight decrease for at least one year [67]. Physical activity is best for weight loss maintenance. Community actions must promote healthy eating and exercise. Urban design, safety, leisure time availability, and economic variables influence these initiatives. Physical exercise has been shown to reduce the risk of cancer, especially renal cell cancer, breast cancer after menopause, endometrial cancer, pancreatic cancer, colon cancer, and oesophageal cancer [68]. These relationships are known to have a direct link to fat. But there are also negative links between exercise and the chance of bladder cancer and stomach cancer. This shows that physical activity has benefits other than preventing obesity. Physical exercise has also been linked to better prognoses and longer lives for people with cancer, especially breast and colon cancer. The effects of physical activity on inflammation, immunity, insulin sensitivity, adiponectin, leptin, and sex steroids are thought to be the reason why it can help avoid and treat cancer [69].

Infectious Agents

Different viruses are involved in infection caused by cancer. Viruses like HCV, HBV, HIV, HPV, EBV causes many types of cancer like liver cancer, leukaemia, sarcoma etc [70, 71].

Modulation of age and cancer

The study revealed that specific lifestyle factors impacted the stability of gene promoter methylation as individuals aged [72]. Individuals who used aspirin or underwent hormonal replacement therapy (HRT) experienced fewer methylation changes than non-users. Conversely, smokers and individuals with a higher BMI exhibited more methylation changes. Interestingly, half of the methylation changes associated with aging were also observed in colorectal cancer (CRC) samples [73]. These changes occurred faster than age-related changes and were frequently found in regions connected to polycomb proteins, which play a role in gene regulation. Significantly, aspirin and HRT were found to reduce the rate of methylation changes in genes associated with cancer; while smoking and high BMI increased the rate. These findings suggest that lifestyle factors, including the use of aspirin, can influence DNA methylation changes in epithelial cells of the colon and potentially affect the development of cancer-related methylation patterns [74].

Fruit and vegetable intakes and prostate cancer risk

The main purpose of this study is to test that as the vegetable and fruit consumption increased it would decrease the risk of cancer formation (breast cancer) in women. In this study 28 women participated. Isolation of DNA from WBCs, 8-OHdG determined by electrochemical detection which is excreted by urine. Pre and post- intervention obtained by HPLC [75]. As higher use of fruit and vegetables are inversely proportional to the cancer risk mostly in women (breast cancer) as well as in animal. Plant food inhibits the cancer risk because mostly plant food possesses the anti-carcinogenic properties. Plant food provides approximately all essential nutrients which have antioxidants properties. These properties help to inhibit the cancer formation (breast cancer) [76, 77]. Interestingly, research has shown that incorporating more vegetables into diet, particularly cruciferous vegetables such as broccoli and cauliflower is associated with a decreased risk of prostate cancer. Men who ingested 28 or more portions of vegetables per week demonstrated a 35% reduced likelihood of developing prostate cancer compared to those who consumed fewer than 14 servings [78]. Moreover, the research indicated that men who consumed a minimum of three servings of cruciferous vegetables per week had a 41%

lower risk than those who consumed less than one serving. Apart from considering overall vegetable consumption, the study also examined the impact of specific nutrients present in vegetables. The study found that men with a daily intake of 2000 micrograms or more of lutein plus zeaxanthin, a particular nutrient, had a lower risk of prostate cancer than those with an infusion of less than 800 micrograms [79]. However, this result was not statistically significant. This study suggests that eating lots of vegetables, especially cruciferous vegetables, may help reduce the risk of prostate cancer in men under 65. However, eating more fruits showed a different strong association. So, including plenty of vegetables in diet, especially ones like broccoli and cauliflower, might be beneficial for preventing prostate cancer [80].

Cancer and young generation

Smoking, drinking, and obesity are discussed as cancer risk factors. Cancer risk factors including smoking, excessive alcohol use, and being overweight are highlighted as areas where a change in lifestyle may have a significant impact. Breast cancer risk variables such as age at menarche, first birth, and balance in women are all changing rapidly in different parts of the world[81]. Tobacco exposure, smoking and second-hand smoke is the biggest cancer risk factor. In industrialized nations, smoking prevalence in males increased and subsequently declined, followed by a similar pattern in tobacco-related mortality [82]. Women follow similar tendencies 10-20 years later. Sub-Saharan Africa has low smoking rates and Western Europe high rates. If low- and middle-income nations replicate affluent countries' cancer rates, future trends will be similar. Based on Scotland's lung cancer pandemic, which peaked 40 years after the smoking outbreak, China's male lung cancer death rate is expected to rise. The rising cancer rate can be attributed to a number of causes, including improved diagnostic tools, better treatment options, and more access to information. Cancers of the UADT, liver, colon, and breast have all been associated to alcohol intake [83]. The rising obesity pandemic is having a major effect on the total cancer burden since it increases the risk for a number of malignancies. The chance of developing breast cancer after menopause is rising in line with changes in women's reproductive habits, such as earlier menarche, later age at first full-term pregnancy, and lower parity. Breast cancer rates are raising because of contemporary lifestyle changes that postpone having children and shorten the nursing time. As a result of all of these causes, cancer rates are rising rapidly over the world, especially in low and middle-income countries [84].

Causes of cancers

There are following common cause of cancer:

- Mostly lung cancer is caused by industrial factor like Asbestos
- Carcinogens (Asbestos) approximately 4 to 11% people died from asbestos in world
- Second hand smoke (approximately 6 to 13% people died)
- Emission of tobacco smoke
- Aromatic hydrocarbons
- Indium phosphate (carcinogen)
- Refractory ceramic fibre [85, 86]

Mechanisms of cancer formation

Research indicates that physical activity or exercise may significantly impact the trajectory of cancer survivorship at various stages. Physical activity has been linked to a decreased risk of developing breast and colon cancer [69]. Although the precise causal mechanism behind this association is not yet fully understood, the observational evidence supporting it is robust and compelling. Apart from its potential for primary cancer prevention, physical activity has demonstrated promise as a valuable intervention during and after cancer treatment [87, 88]. Despite advancements in cancer therapies, many treatments still lead to adverse symptoms and side effects. However, evidence indicates that physical activity can help alleviate many of these treatment-related symptoms. Engaging in physical activity during and after cancer treatment improves multiple physiological systems, enhancing physiological and psychosocial outcomes [89].

The positive impact of physical activity throughout the cancer journey underscores its potential as a complementary therapy. However, several research gaps still need to be addressed. One significant research gap is determining the optimal amount of physical activity required to maximize the reduction in the risk of developing cancer. Understanding the specific duration, intensity, and frequency of exercise needed to attain optimal benefits is crucial. Similarly, investigating the ideal amount of physical activity required to improve specific physiological systems or alleviate treatment-specific side effects is another vital area of research. We can enhance the benefits of physical activity interventions by identifying the most effective exercise regimens tailored to different cancer types, treatment stages, and individual patient characteristics [90]. Moreover, to establish cancer rehabilitation as a standard element of cancer care covered by third-party providers, it is crucial to substantiate the safety and cost-effectiveness of physical medicine and

rehabilitation interventions. It is imperative to generate strong evidence demonstrating these interventions' safety, effectiveness, and economic value, thereby promoting their widespread adoption and ensuring their accessibility.

The available evidence strongly supports the significance of physical activity in cancer survivorship. Engaging in physical activity has been linked to a decreased risk of cancer development and has shown promising results in alleviating treatment-related symptoms. However, additional research is required to determine the ideal exercise dosage for risk reduction and symptom management. Moreover, it is vital to establish the safety and cost-effectiveness of physical medicine and rehabilitation interventions to ensure their integration into comprehensive cancer care. By addressing these research gaps, we can fully leverage the potential of physical activity to enhance outcomes across the entire cancer journey [91]. Numerous studies have extensively explored the correlation between physical activity and cancer risk. Breast and colon cancers have amassed compelling evidence linking them to certain risk factors. There is probable evidence indicating a connection between risk factors and prostate cancer, while the evidence is considered possible for lung and endometrial cancers. However, the available evidence is currently insufficient to establish a strong association with other types of cancer. However, further investigation is needed to thoroughly analyse various factors that may influence these findings, such as the intensity of physical activity and other relevant variables. Physical activity can potentially reduce cancer risk through multiple mechanisms, including hormonal regulation, metabolic changes, improved immune function, and maintenance of a healthy weight [92]. Excess weight, especially in specific body areas, has been associated with increased cancer risk. Drawing from the existing evidence, several public health organizations recommend engaging in at least 30 minutes of moderate-to-vigorous physical activity on most days of the week for cancer prevention. Additionally, beyond its preventive effects, exercise has positively impacted various aspects of the cancer experience, including early detection, coping, rehabilitation, and survival rates following diagnosis [93].

Diet, nutrition and the prevention of cancer

More chances of industrial cancer have those of people who have low immunity level such as aged people and very young child or those people who have acute diseases. To prevent lung cancer, we should keep away such people from hazardous place. E.U. banned uses of all type of asbestos in industries [94]. In 1981, Doll and Peto, they give the percentage of cancer death about 30 to 40% due to Diet in United State of America [95]. Diet plays an impotent role in CRC about 70% people died due to colorectal cancer [96]. Many carcinogens enter to our body along with food or vegetables. Carcinogens like asbestos, nitrates, pesticide, and dioxin etc. CRC is most common cancer in men and women as well. Approximately 7.4 million/ year cases in men registered while approximately 6.1 million cases in women registered every year in the world [97].

Since the 1950s, researchers have been investigating the link between nutrition and cancer. Investigations in laboratories and animal studies were used in early research. Preliminary dietary guidelines were released in the 1980s. In the late 1990s, a review panel organized by the World Cancer Research Fund and the American Institute for Cancer Research published an authoritative assessment on Diet, nutrition, and cancer prevention. The accuracy of estimating people's nutritional consumption is restricted, and retrospective approaches might be biased. The research is additionally complicated by confounding relationships between dietary and non-dietary cancer risk factors. Initial findings showed that eating more vegetables and fruits might lower cancer risks, but later research has not consistently confirmed this. Similarly, based on more recent prospective studies, early evidence associating excessive fat diets with cancer risk has been lowered to limited or suggestive strength. The Women's Health Initiative, a large scale randomized study including over 48,000 postmenopausal women, found no obvious impact of dietary fat reduction on cancer incidence or death [98].

Research consistently demonstrates that being overweight or obese significantly elevates the risk of several types of cancer. These include oesophageal adeno carcinoma, colorectal cancer, postmenopausal breast cancer, endometrial cancer, and kidney cancer. Therefore, striving to maintain a healthy weight is crucial for reducing the likelihood of developing these cancers. To mitigate these risks, avoiding weight gain in adulthood is advisable. The consumption of alcohol has been linked to an increased risk of several cancers, such as those affecting the oral cavity, pharynx, oesophagus, liver, and a slightly higher risk of breast cancer. To minimize these risks, it is recommended that alcohol intake should not exceed two units per day. Aflatoxin, a substance found in certain foods, has been associated with liver cancer, although its significance without hepatitis virus infections remains unclear[99]. To reduce the risk, minimizing exposure to aflatoxin in foods is advisable. Chinese-style salted fish, particularly during

childhood, has increased the risk of nasopharyngeal cancer. It is recommended to consume this type of fish in moderation. Including fruits and vegetables in the daily diet is believed to lower the risk of oral cavity, oesophageal, stomach, and colorectal cancers. It is advised to consume a total of 400 grams of fruits and vegetables each day. Preserved and red meat is likely to increase the risk of colorectal cancer [100]. If ingested, it is recommended to do so in moderation. High salt intake and the consumption of salt preserved foods are associated with an increased risk of stomach cancer. Therefore, it is advised to moderate the overall consumption of these foods. Consumption of scorching hot drinks and nutrition has increased the risk of oral cavity, pharyngeal, and oesophageal cancers. It is recommended to avoid consuming drinks and food when they are excessively hot. Regular physical activity, which helps to expend energy, has been associated with a reduced risk of colorectal and possibly breast cancer. Regular physical activity is encouraged to lower the risk of these cancers. It is important to note that these recommendations are based on current scientific findings and are subjected to ongoing research. Seeking advice from healthcare experts or licensed dietitians can offer tailored guidance and counsel, taking into account personal health considerations and medical background [101]. Inpatient drug addiction therapy improves smoking cessation without affecting abstinence. The most effective strategies for smoking cessation in alcohol dependent persons are not clear, but behavioural therapy that is aligned with counselling approaches for alcohol dependence has shown to be effective [102].

A comprehensive guide to WHO study

The WHO study reveals that a significant portion of cancer deaths globally can be attributed to lifestyle factors that can be modified [103]. These factors include smoking and alcohol use, and they have an impact on both high- and low-income countries. The prevalence of HPV infection is a significant contributor to the development of cervical cancer. However, there are ongoing efforts in certain countries to introduce vaccines as a preventive measure. The WHO report does not cover other lifestyle factors like occupational settings, U.V. light exposure, tobacco smoking, dietary factors, or radiation exposure [104].

Precautions by WHO

A PAL of 1.4 or below indicates sedentary behaviour, whereas the usual range indicates healthy adult development. Reducing intake of high-calorie items like fast food and red meat is recommended. Cereals and pulses, on the other hand, have a low energy density and encourage a healthy weight, whereas fast food is both high in energy density and low in energy density. Children and pregnant women should not drink alcohol, and nursing is safe for both the mother and the kid. Those who have been diagnosed with cancer and are still alive after treatment and/or the disease's progression are considered cancer survivors [105].

The American Cancer Society provides guidelines and recommendations for cancer prevention and treatment, emphasizing the importance of maintaining a healthy lifestyle. This includes choosing nutritious foods, limiting processed and red meats and sugary drinks, and maintaining a balanced diet low in saturated and trans fats. Regular physical activity is also encouraged, with a minimum of 150 minutes of moderate-intensity exercise or 75 minutes of vigorous-intensity exercise per week [106]. Strength training exercises should be incorporated at least two days per week. Following these guidelines can reduce the risk of developing certain types of cancer. Other factors that can impact cancer risk include postmenopausal hormone therapy, weight gain, alcohol intake, and physical activity. For specific types of cancer, such as breast cancer, a diet abundant in vegetables, fruits, poultry, fish, and low-fat dairy products can lower the risk [107].

For colorectal cancer, avoiding red and processed meats and consuming diets rich in vegetables, fruits, and whole grains can decrease the risk. Obesity is strongly linked to endometrial cancer, and regular physical activity is inversely associated with the risk. Kidney cancer risk can be reduced by maintaining a healthy weight and avoiding tobacco use. Lung cancer risk is primarily attributed to tobacco smoking and radon exposure, but physical activity and a diet rich in vegetables and fruits can help reduce the risk. Ovarian cancer risk is associated with obesity, and some evidence suggests that a higher intake of vegetables, fruits, soy foods, and tea may reduce the risk [108]. Pancreatic cancer risk can be decreased by avoiding tobacco, maintaining a healthy weight, and adopting a healthy diet. Prostate cancer risk is increased by being overweight or obese, but regular physical activity can help reduce the risk. Alcohol consumption increases certain cancer risks, while antioxidants from fruits and vegetables may lower the risk. Other dietary factors, such as calcium intake, dairy consumption, and the Impact of vitamin C, require further investigation [109]. Overall, following a diverse diet rich in fruits and vegetables, engaging in regular physical activity, and maintaining a healthy weight are recommended for reducing cancer risk [110].

Effective strategies for quitting smoking and alcohol consumption

Nicotine replacement therapy, such as nicotine gum and patches, has been used in smoking cessation studies of alcohol dependent persons, but the effectiveness may be related to adequate nicotine replacement. Bupropion, an anti-depressant, is another pharmacological agent that can be used for smoking cessation in alcohol dependent persons. Antidepressant bupropion is effective for smoking cessation because it blocks the reuptake of dopamine and noradrenaline. Competitively inhibits nicotinic acetylcholine receptors [111]. Studies on smoking cessation, especially among alcoholics, have proved its efficacy. The principal mechanism of action for smoking cessation is not bupropion's antidepressant effects. Nortriptyline, another antidepressant, has been examined for smoking cessation but has higher negative effects. Heavy smokers may benefit from nicotine replacement therapy and bupropion. Early smoking cessation may improve alcohol therapy. Naltrexone, an opioid antagonist, has reduced smoking but not smoking cessation [4].

Conclusion

Cancer development is a complex, multistep process driven by genetic instability, metabolic reprogramming, and exposure to modifiable and non-modifiable risk factors. This review highlights the critical role of lifestyle and environmental factors, including tobacco use, alcohol consumption, poor dietary habits, physical inactivity, environmental pollution, and metabolic dysregulation, in increasing cancer risk. Importantly, evidence indicates that positive lifestyle modifications such as adherence to a balanced diet, calorie control, regular physical activity, maintenance of healthy body weight, and avoidance of harmful behaviors can counteract metabolic alterations associated with tumorigenesis and significantly reduce cancer incidence. Integrating lifestyle-based prevention strategies with public health policies and early intervention programs is essential for reducing the global cancer burden and improving long-term health outcomes.

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References

1. Engelmann, K., Shen, H., and Finn, O.J., Supplementary Figure 1 from MCF7 Side Population Cells with Characteristics of Cancer Stem/Progenitor Cells Express the Tumor Antigen MUC1. **2023**, American Association for Cancer Research (AACR).
2. Kim, J. and Keegan, T.H., Characterizing Disparities in Risky Alcohol Use, Tobacco Smoking, E-Cigarette Use and Physical Inactivity Among Cancer Survivors in the United States. **2022**, Research Square Platform LLC.
3. Khan N, A.F., Mukhtar H. Lifestyle as risk factor for cancer: Evidence from human studies. Cancer letters. 2010 Jul 28 and 293(2):133-43.
4. Begum, S., Yada, A., and Lorencatto, F. How Has Intervention Fidelity Been Assessed in Smoking Cessation Interventions? A Systematic Review. Journal of smoking cessation. **2021**. 2021: 6641208-6641208 10.1155/2021/6641208.
5. Mathur, S. and Levy, M. Background: cigarette smoking is the greatest known risk factor for lung cancer, and people with different smoking status may process risk information differently. While psychological distress has been linked with smoking status, little is known about the. Epidemiology, Biostatistics, and Public Health. **2022**. 10(2) 10.2427/8839.
6. naseem, r., The Impact of Lifestyle Choices on Cancer Risk Reduction. **2023**, Center for Open Science.
7. Kushi, L.H., Doyle, C., et al. American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. CA: a cancer journal for clinicians. **2012**. 62(1): 30-67.
8. Justice, in *Social Choice*. **1992**, Cambridge University Press. p. 123-132.
9. Khan, N., Afaq, F., and Mukhtar, H. Lifestyle as risk factor for cancer: Evidence from human studies. Cancer letters. **2010**. 293(2): 133-143.
10. Anand, P., Kunnumakara, A.B., et al. Cancer is a preventable disease that requires major lifestyle changes. Pharmaceutical research. **2008**. 25: 2097-2116.
11. Aleksandrova, K., Pischon, T., et al. Combined impact of healthy lifestyle factors on colorectal cancer: a large European cohort study. BMC medicine. **2014**. 12(1): 1-15.
12. Calle, E.E. and Thun, M.J. Obesity and cancer. Oncogene. **2004**. 23(38): 6365-6378.
13. Parkin, D.M., Boyd, L., and Walker, L. 16. The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010. British journal of cancer. **2011**. 105(2): S77-S81.

14. Hylen, S.E., Marriage, Divorce, and Widowhood, in *Oxford Scholarship Online*. **2018**, Oxford University Press.
15. Tonin, S., Alberini, A., and Turvani, M. The Value of Reducing Cancer Risks at Contaminated Sites: Are More Knowledgeable People Willing to Pay More? Risk Analysis. **2011**. 32(7): 1157-1182 10.1111/j.1539-6924.2011.01730.x.
16. Hemminki, K. and Li, X. Lifestyle and cancer: effect of widowhood and divorce. Cancer Epidemiology Biomarkers & Prevention. **2003**. 12(9): 899-904.
17. Peers, F., Bosch, X., et al. Aflatoxin exposure, hepatitis b virus infection and liver cancer in swaziland. International Journal of Cancer. **1987**. 39(5): 545-553 10.1002/ijc.2910390502.
18. Key, T.J., Schatzkin, A., et al. Diet, nutrition and the prevention of cancer. Public health nutrition. **2004**. 7(1a): 187-200.
19. Choi, J.-W. and Hua, T.N. Impact of lifestyle behaviors on cancer risk and prevention. Journal of Lifestyle Medicine. **2021**. 11(1): 1.
20. Cohen, J.H., Kristal, A.R., and Stanford, J.L. Fruit and vegetable intakes and prostate cancer risk. Journal of the National Cancer Institute. **2000**. 92(1): 61-68.
21. Noreen, F., Röösli, M., et al. Modulation of age-and cancer-associated DNA methylation change in the healthy colon by aspirin and lifestyle. JNCI: Journal of the National Cancer Institute. **2014**. 106(7): dju161.
22. Friedenreich, C.M. and Orenstein, M.R. Physical activity and cancer prevention: etiologic evidence and biological mechanisms. The Journal of nutrition. **2002**. 132(11): 3456S-3464S.
23. Transfer of Poly(methyl methacrylate) Nanoparticles from Parents to Offspring and the Protection Mechanism in Two Marine Invertebrates. American Chemical Society (ACS).
24. Kaushal, M., Mishra, A.K., et al. Betel quid chewing as an environmental risk factor for breast cancer. Mutation Research/Genetic Toxicology and Environmental Mutagenesis. **2010**. 703(2): 143-148 10.1016/j.mrgentox.2010.08.011.
25. Muturi, N. Alcohol consumption and reproductive health risks in rural Central Kenya. Sexual & Reproductive Healthcare. **2014**. 5(2): 41-46 10.1016/j.srhc.2014.01.002.
26. Rayan, R.A., Knowledge of Preventable Risk Factors and Beliefs About Cancer A Cross-Sectional Study. **2020**, Center for Open Science.
27. Printz, C. Four in 10 cancer deaths attributed to smoking in South, Appalachia. Cancer. **2021**. 127(15): 2607-2607 10.1002/cncr.33758.
28. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. BMJ. **2014**. 349(sep03 18): g5472-g5472 10.1136/bmj.g5472.
29. Usher-Smith, J., A randomised controlled trial of the effect of providing online risk information and lifestyle advice for the most common preventable cancers, in http://isrctn.com/. **2018**, Springer Science and Business Media LLC.
30. Hicks, J., Health Workforce Interventions in Low-and-Middle-Income-Countries (LMICs). **2022**, Institute of Development Studies.
31. All about prostate cancer All about prostate cancer. Nursing Standard. **2001**. 15(36): 29-29 10.7748/ns2001.05.15.36.29.b315.
32. Cummings, K.M. Abstract ED05-03: Global tobacco control: Preventing 10 million cancer deaths by 2030. Cancer Prevention Research. **2010**. 3(1_Supplement): ED05-03-ED05-03 10.1158/1940-6207.prev-09-ed05-03.
33. Printz, C. Smokeless tobacco product may increase mortality rate in patients with prostate cancer. Cancer. **2017**. 123(2): 190-190 10.1002/cncr.30523.
34. Anderson, B.A., 7. Marriage , Family, and Fertility Data in Russian and Soviet Censuses, in *Research Guide to the Russian and Soviet Censuses*. **2017**, Cornell University Press. p. 131-154.
35. Rayhill, S., Dick, A., et al. For Pediatric Patients Listed for Simultaneous Liver-Kidney Transplantation, Those Who Received Both a Liver and a Kidney Transplant Had Significantly Better Graft and Patient Survival Than Those Who Received Only a Liver Transplant. Transplantation. **2014**. 98: 756-757 10.1097/00007890-201407151-02583.
36. Lindqvist, P.G., Boström, L., et al., Appendectomy during pregnancy: rates, safety, and outcomes over a five-year period. A hospital-based follow-up study. **2022**, Authorea, Inc.
37. Mohammed, E.Z., Jameel, N.G.M., and Shukr, A., Forecasting Cancer Incidences Using Machine Learning Techniques Based on Annual Reported Time Series Database. **2023**, Elsevier BV.
38. Rettew, D., Pick Them Up or Let Them Cry (for a While)?, in *Parenting Made Complicated*. **2021**, Oxford University Press. p. 57-75.
39. Marriage rates declined while divorce rates stabilised over the last decades. **2019**, Organisation for Economic Co-Operation and Development (OECD).
40. Hemminki, K., Dong, C., and Frisch, M. Tonsillar and other upper aerodigestive tract cancers among cervical cancer patients and their husbands. European Journal of Cancer Prevention. **2000**. 9(6): 433-437 10.1097/00008469-200012000-00010.
41. Adolescent pregnancies stem from discriminatory social institutions such as child marriage among girls. **2021**, Organisation for Economic Co-Operation and Development (OECD).
42. The use of tobacco is a risk factor for poor outcomes and readmissions after shoulder arthroplasty, in *OrthoMedia*. **2022**, British Editorial Society of Bone & Joint Surgery.
43. 90-day delinquency rate (%) and GDP in Canada, 2007-16. **2018**, Organisation for Economic Co-Operation and Development (OECD).
44. Lubin, J.H., Albanes, D., et al. Greater Coronary Heart Disease Risk With Lower Intensity and Longer Duration

Smoking Compared With Higher Intensity and Shorter Duration Smoking: Congruent Results Across Diverse Cohorts. Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco. **2017**. 19(7): 817-825 10.1093/ntr/ntw290.
45. Lin, C.-Y., THE PREVALENCE AND RISK OF SECOND PRIMARY MALIGNANCIES AFTER DIFFERENT STAGES OF BREAST CANCER WOMEN. **2019**, Morressier.
46. McCambridge, J. and Jenkins, R.J. Do brief interventions which target alcohol consumption also reduce cigarette smoking? Drug and Alcohol Dependence. **2008**. 96(3): 263-270 10.1016/j.drugalcdep.2008.03.011.
47. Older smokers more likely to become frail than non-smokers. Clinical Pharmacist. **2017** 10.1211/cp.2017.20203679.
48. Windmill, K.F. Localization of N-Acetyltransferases NAT1 and NAT2 in Human Tissues. Toxicological Sciences. **2000**. 54(1): 19-29 10.1093/toxsci/54.1.19.
49. Weinberger, A.H., Wyka, K., and Goodwin, R.D. Impact of cannabis legalization in the United States on trends in cannabis use and daily cannabis use among individuals who smoke cigarettes. Drug and Alcohol Dependence. **2022**. 238: 109563 10.1016/j.drugalcdep.2022.109563.
50. Guven, G.S., Guven, M., et al. Enhanced Sensitivity to Oxidant-Induced Micronucleus Frequency in Elderly Individuals Is Not Associated with Glutathione S-Transferase M1 (GSTM1) Null Genotype in Lymphocytes. Gerontology. **2004**. 51(1): 29-33 10.1159/000081431.
51. Baldrich, J.J., About the Author, in *Smoker beyond the Sea*. **2022**, University Press of Mississippi. p. 272-272.
52. He, F., Sha, Y., and Wang, B. Relationship between alcohol consumption and the risks of liver cancer, esophageal cancer, and gastric cancer in China: Meta-analysis based on case-control studies. Medicine. **2021**. 100(33): e26982-e26982 10.1097/MD.0000000000026982.
53. Guillén-Mancina, E., Calderón-Montaño, J.M., and López-Lázaro, M., A Simple Way to Reduce the Risk of Cancer of the Oral Cavity, Pharynx, Larynx and Esophagus in Alcohol Users. **2017**, MDPI AG.
54. Zakhari, S. and Hoek, J.B. Epidemiology of Moderate Alcohol Consumption and Breast Cancer: Association or Causation? Cancers. **2018**. 10(10): 349 10.3390/cancers10100349.
55. Jayasekara, H., MacInnis, R.J., et al. Long-Term Alcohol Consumption and Breast, Upper Aero-Digestive Tract and Colorectal Cancer Risk: A Systematic Review and Meta-Analysis. Alcohol and Alcoholism. **2015**. 51(3): 315-330 10.1093/alcalc/agv110.
56. Brown, K.A. and Simpson, E.R., The Link Between Obesity and Breast Cancer Risk: Epidemiological Evidence, in *Obesity and Breast Cancer*. **2013**, Springer New York. p. 5-10.
57. Men on high doses of amiodarone show increased chance of developing cancer. Nursing Standard. **2013**. 27(35): 14-14 10.7748/ns2013.05.27.35.14.s20.
58. Bhattacharya, N. and Sengupta, P., Body Mass Index (BMI) and Anthropometric Measurement of the Developing Fetus, in *Body-mass Index and Health*. **2019**, IntechOpen.
59. Imada, K., Shiota, M., et al. FOXO3a Expression Regulated by ERK Signaling is Inversely Correlated With Y-Box Binding Protein-1 Expression in Prostate Cancer. The Prostate. **2016**. 77(2): 145-153 10.1002/pros.23254.
60. Speakman, J.R. and Goran, M.I. Tissue-Specificity and Ethnic Diversity in Obesity-Related Risk of Cancer May Be Explained by Variability in Insulin Response and Insulin Signaling Pathways. Obesity. **2010**. 18(6): 1071-1078 10.1038/oby.2010.16.
61. Rehm, J. Light or moderate drinking is linked to alcohol related cancers, including breast cancer. BMJ. **2015**: h4400 10.1136/bmj.h4400.
62. Arthi, G., Sadeesh, T., and Raghuram, K. Effect of Height, Weight and BMI on Foot Postures of Young Adult Individuals. JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH. **2018** 10.7860/jcdr/2018/30914.11970.
63. Borchert, D.M., Pink hibiscus mealybug — how our regulatory response and understanding of PHM has changed over time, in *2016 International Congress of Entomology*. **2016**, Entomological Society of America.
64. Moltrer, M., Pala, L., et al. Body mass index (BMI), waist circumference (WC), waist-to-height ratio (WHtR) e waist body mass index (wBMI): Which is better? Endocrine. **2022**. 76(3): 578-583 10.1007/s12020-022-03030-x.
65. Raskiliene, A., Smalinskiene, A., et al. Associations of MC4R, LEP, and LEPR Polymorphisms with Obesity-Related Parameters in Childhood and Adulthood. Genes. **2021**. 12(6): 949 10.3390/genes12060949.
66. Davis, D., Brown, W.J., et al. Long-Term Weight Gain and Risk of Overweight in Parous and Nulliparous Women. Obesity. **2018**. 26(6): 1072-1077 10.1002/oby.22174.
67. Lillis, J., Thomas, J.G., et al. Internal disinhibition predicts 5-year weight regain in the National Weight Control Registry (NWCR). Obesity science & practice. **2016**. 2(1): 83-87 10.1002/osp4.22.
68. Gynaecological Cancers Risk: Breast Cancer, Ovarian Cancer and Endometrial Cancer. **2022**, MDPI.
69. Al-Mhanna, S.B., Ghazali, W.S.W., et al. Effectiveness of physical activity on immunity markers and quality of life in cancer patient: a systematic review. PeerJ. **2022**. 10: e13664.
70. Ahmed, N., Abusalah, M.A.H.A., et al. Updates on Epstein–Barr Virus (EBV)-Associated Nasopharyngeal Carcinoma: Emphasis on the Latent Gene Products of EBV. Medicina. **2022**. 59(1): 2.
71. Ahmed, N., Rabaan, A.A., et al. Immunoinformatic Execution and Design of an Anti-Epstein–Barr Virus Vaccine with Multiple Epitopes Triggering Innate and Adaptive Immune Responses. Microorganisms. **2023**. 11(10): 2448.
72. COX-2 Gene Expression in Colon Cancer Tissue Related

to Regulating Factors and Promoter Methylation Status, in *Specific Gene Expression and Epigenetics*. **2014**, Apple Academic Press. p. 59-80.
73. Young, G.P. Changes in cancer patterns from widespread testing - colorectal cancer (CRC) screening. Pathology. **2014**. 46: S13 10.1097/01.pat.0000443438.90561.10.
74. Carbon Nanotube- and Asbestos-Induced DNA and RNA Methylation Changes in Bronchial Epithelial Cells. American Chemical Society (ACS).
75. Laws, G.M. and Adams, S.P. Measurement of 8-OHdG in DNA by HPLC/ECD: The Importance of DNA Purity. BioTechniques. **1996**. 20(1): 36-38 10.2144/96201bm06.
76. Ali, A., Alghamdi, M., et al. Exploring AI Approaches for Breast Cancer Detection and Diagnosis: A Review Article. Breast Cancer: Targets and Therapy. **2025**: 927-947.
77. Khalaf, T.K., Ismail, N., et al. Pereskia bleo augments NK cell cytotoxicity against triple-negative breast cancer cells (MDA-MB-231). PeerJ. **2024**. 12: e18420.
78. Number of Years When Consumed More than 2 Drinks per Day for Men and More than 1 Drink per Day for Women, in *Definitions*. **2020**, Qeios.
79. Exposure to air pollution remains widespread. **2021**, Organisation for Economic Co-Operation and Development (OECD).
80. Bhattacharjee, P. and Singhal, R.S., Broccoli and Cauliflower, in *Handbook of Vegetables and Vegetable Processing*. **2018**, John Wiley & Sons, Ltd. p. 535-558.
81. Li, C.I., Littman, A.J., and White, E. Relationship between Age Maximum Height Is Attained, Age at Menarche, and Age at First Full-Term Birth and Breast Cancer Risk. Cancer Epidemiology, Biomarkers & Prevention. **2007**. 16(10): 2144-2149 10.1158/1055-9965.epi-07-0242.
82. Kulik, M.C. and Glantz, S.A. Similar softening across different racial and ethnic groups of smokers in California as smoking prevalence declined. Preventive medicine. **2019**. 120: 144-149 10.1016/j.ypmed.2019.01.020.
83. Balaam, S.M., Alcohol Use and Associated Health Behaviours of Women who have Been Treated for Breast Cancer. Queensland University of Technology.
84. Tekin, M., Under-Five Mortality Causes and Prevention, in *Mortality Rates in Middle and Low-Income Countries*. **2022**, IntechOpen.
85. Ahmed, N., Abusalah, M.A.H.A., et al. The Microbial Sources of Bioactive Compounds: Potential Anticancer Therapeutic Options. Nano Life. **2025**. 15(04): 2430007.
86. Ahmed, N., Abusalah, M.A.H.A., et al. Carbon-based Nanomaterials as Multifunctional Particles for Cancer Diagnosis and Treatment. Nano Life. **2025**. 15(04): 2430005.
87. Ahmad, S., Lambuk, L., et al. Efficacy and safety of nab-paclitaxel in metastatic gastric cancer: a meta-analysis. Nanomedicine. **2023**. 18(24): 1733-1744.
88. Mohd Salim, N.H., Mussa, A., et al. The Immunosuppressive Effect of TNFR2 Expression in the Colorectal Cancer Microenvironment. Biomedicines. **2023**. 11(1): 173.
89. Hyatt, J.-P.K. MOTS-c increases in skeletal muscle following long-term physical activity and improves acute exercise performance after a single dose. Physiological reports. **2022**. 10(13): e15377-e15377 10.14814/phy2.15377.
90. Perperidi, M., Saliari, D., et al. Identifying the effective behaviour change techniques in nutrition and physical activity interventions for the treatment of overweight/obesity in post-treatment breast cancer survivors: a systematic review. Cancer causes & control : CCC. **2023**. 34(8): 683-703 10.1007/s10552-023-01707-w.
91. Arikan, A., Solve Challenges Across the Entire Customer Journey, in *Customer Experience Analytics*. **2023**, Routledge. p. 246-288.
92. Physiological Changes in Multiple Organ Systems Through Aging, in *Prevention and Management of Cardiovascular and Metabolic Disease*. **2023**, Wiley. p. 117-132.
93. berkat, w., The Role of Early Detection in Improving Cancer Survival Rates. **2023**, Center for Open Science.
94. Levin, L., Properties and Uses of Asbestos, in *Asbestos: The Hazardous Fiber*. **2018**, CRC Press. p. 27-43.
95. Peto, J. and Doll, R. Prof. Peto and Sir Richard Doll reply. British Journal of Cancer. **1986**. 54(6): 1020-1021 10.1038/bjc.1986.276.
96. Gut Microbiome Plays Important Role in Colorectal Cancer Growth, in *Default Digital Object Group*. **2022**, American Society of Clinical Oncology (ASCO).
97. Harmful use of alcohol kills more than 3 million people each year, most of them men, in *Human Rights Documents Online*. Brill.
98. Tinker, L.F. Low-Fat Dietary Pattern and Risk of Treated Diabetes Mellitus in Postmenopausal Women<subtitle>The Women's Health Initiative Randomized Controlled Dietary Modification Trial</subtitle&gt. Archives of Internal Medicine. **2008**. 168(14): 1500 10.1001/archinte.168.14.1500.
99. Although it remains under-researched, unconditional positive regard has been reconsidered and re-evaluated, in *Person-Centred Therapy*. **2015**, Routledge. p. 110-111.
100. Environmentally concerned respondents are less likely to consume red meat several times a week. **2023**, Organisation for Economic Co-Operation and Development (OECD).
101. Stephenson, J. Experts Offer Advice to Shore Up Confidence in COVID-19 Vaccines. JAMA Health Forum. **2021**. 2(2): e210158 10.1001/jamahealthforum.2021.0158.
102. Brown, J., Kotz, D., et al. How effective and cost-effective was the national mass media smoking cessation campaign 'Stoptober'? Drug and alcohol dependence. **2014**. 135: 52-58 10.1016/j.drugalcdep.2013.11.003.
103. Podcast: How Oncologists Can Address Lifestyle Factors in Cancer Care, in *Default Digital Object Group*. **2021**,

American Society of Clinical Oncology (ASCO).
104. Mlinarić, M., Kastaun, S., and Kotz, D. Exposure to Tobacco Smoking in Vehicles, Indoor, and Outdoor Settings in Germany: Prevalence and Associated Factors. International journal of environmental research and public health. **2022**. 19(7): 4051 10.3390/ijerph19074051.
105. Young cancer survivors need support to make healthy lifestyle choices. Nursing Standard. **2012**. 26(44): 14-14 10.7748/ns.26.44.14.s17.
106. Cruz Maldonado, G., The influence of regular physical activity on affective and mood state response to acute moderate intensity exercise in adults with major depressive disorder. Iowa State University.
107. Printz, C. Low-fat diet in postmenopausal women can lower risk of death from invasive breast cancers. Cancer. **2016**. 122(20): 3101-3101 10.1002/cncr.30359.
108. Mayor, S. Threshold for surgery to reduce ovarian cancer risk should be lowered, suggests study. BMJ. **2016**: i3559 10.1136/bmj.i3559.
109. Mussa, A., Mohd Idris, R.A., et al. High-dose vitamin C for cancer therapy. Pharmaceuticals. **2022**. 15(6): 711.
110. McTiernan, A. Physical Activity, Weight, Diet, and Breast Cancer Risk Reduction. Archives of Internal Medicine. **2010**. 170(20) 10.1001/archinternmed.2010.416.
111. Bupropion inhibits human α3β4 nicotinic acetylcholine receptors by interacting with luminal and non-luminal sites. Neurotransmitter. **2018**. 5 10.14800/nt.1631.