

# The Role of Nutrition and Exercise in the Prevention of the Onset of Cancer

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#### Abstract

With cancer being the second leading cause of death in the United States, it is imperative to take as many precautions as possible to its onset. Studies have shown that sound nutrition practices and regular exercise create changes in the body that facilitate the prevention of cancer. This paper presents a comprehensive review of these topics, as well as some practical tips that will enhance the health of an individual and help prevent the onset of cancer.

Keywords: Cancer; Nutrition; Exercise

## Introduction

Cancer results from a process known as carcinogenesis, where cancer cells form through the transformation of normal cells [1,2]. Carcinogenesis can result from a variety of abnormalities in the cellular environment. Cellular processes are very specific in regards to environment and molecules; therefore, miniscule alterations to the environment can be detrimental for the cell and, ultimately, the human being. Carcinogens spur normal cells to undergo a series of changes that lead to uncontrolled cell division, a characteristic of cancer cells. There are many mutations that result from carcinogens that play a role in the formation of cancer cells. However, individuals have the ability to protect and preserve their normal-functioning cells through proper nutrition and exercise.

With cancer being the second leading cause of death in the United States, it is imperative to take as many precautions as possible to its onset. Studies have shown that sound nutrition practices and regular exercise create changes in the body that facilitate the prevention of cancer. In terms of diet, eating fruits, vegetables, and whole grains helps to protect the body from harmful reactive species [1]. Exercise changes levels of sexual hormones, pulmonary function, metabolic hormones, growth factors, and immune function, in addition to controlling one's weight. Thirty-five percent of cancer cases have been attributed to inactivity and an unhealthy diet [3].

#### Nutrition

According to research, changes in dietary behavior are a practical strategy for reducing the risk of the onset of cancer due to the nutrients provided from sound nutrition. These nutrients combat mutations within the cellular environment [1,2,4]. In a study conducted by Block, et al., the risk of cancer was 2-fold higher in persons with a low intake of fruits and vegetables than in those with a high intake for cancer of the lung, colon, breast, cervix, esophagus, oral cavity, stomach, bladder, pancreas, and ovary [1].

## Phytochemicals

An individual's diet plays an essential role in preventing or facilitating cancer. Research has shown that with the correct diet, an individual can use the nutrients they consume to fight the onset of diseases including cancer. Everyone has heard that fruits, vegetables, and whole wheat are beneficial for health, but why? These foods all contain phytochemicals. Phyto means plant in Greek; therefore, phytochemicals are chemicals that are ingested with plant based foods. Increasing the intake of phytochemicals is one way to decrease cancer risk. One reason phytochemicals are important is because they play an antioxidative role to help combat the "the oxidative stress induced by free radicals [which] is involved in the etiology of a wide range of chronic diseases" [1].

Oxidative stress is the result of oxidative agents gaining a much greater concentration than antioxidative agents [1,2,5-7]. Reactive oxidative species (ROS) are normal in the body; they are a result of normal cell function. However, exogenous sources of ROS, more commonly known as carcinogens, have the potential to produce detrimental cellular mutations [1,2]. Carcinogens include, but are not limited to, pollutants, smoke, and radiation. When in low concentrations, ROS are not harmful to the function of a cell; when concentrations rise, ROS cause mutations to components of the normal cell [2]. When these mutations are abundant, the chance of carcinogenesis increases [2]. To help combat the higher levels of ROS, people must consume antioxidants. One source of antioxidants is phytochemicals. They present the body with many antioxidants to lower the concentration of ROS in the body thus lowering the chances of cell mutation. The presence of antioxidants, resulting from consuming foods with phytochemicals, helps to block the harmful effects of elevated levels of ROS [2].

## Isolated phytochemicals

Phytochemical supplements are widely available; however, they are not as effective as consuming the raw form. The preventative effects noticed from consuming fruits, vegetables, and wheat were not found when the supplements were studied individually. Consensus on why this occurs is lacking as the phytochemical loses its bioactivity when isolated [1]. Many researchers assert that the combination of phytochemicals in the foods we consume cause interactions that reverse the cell's destination of carcinogenesis. Therefore, extracts from fruits and vegetables provide the body with the necessary nutrients to induce antioxidative effects.

## **Red meats**

Increased phytochemical consumption is necessary for preventing the onset of cancer, but there are many foods one should avoid in order to lower their risk of cancer. Red meats show a positive correlation with the onset of colorectal, lung, esophageal, and liver cancers [5]. One investigation argues that consumption of red meat directly relates to an increase in colorectal, lung, esophagus, and liver cancers [5]. This protein provides the body with a source of heme iron. The presence of excess iron increases oxidative stress in the cellular environment by increasing the endogenous formation of mutagenic N-nitroso compounds [5]. As discussed previously, oxidative stress must be combatted with antioxidants; when this does not occur, various mutations often lead to chronic disease such as cancer. Therefore, the consumption of red meat enables carcinogenesis by inducing oxidative stress.

## **Processed meats**

Processed meat is harmful due to a similar mechanism as red meat. Through preservation methods, processed meats are an exogenous source of N-nitroso compounds because the food industry uses nitrite compounds [5]. Therefore, processed meats also induced oxidative stress by the introduction of N-nitroso compounds into the cellular environment. Due to a slightly different mechanism, nitrite preserved meats have only been proved to be linked to colorectal and lung cancers [5].

## Alcohol

Moderate consumption of alcohol is widely deemed as beneficial to health. However, increased alcohol consumption is related to the increased risk of occurrence of malignant tumors of the oral cavity, pharynx, larynx, esophagus, liver, colorectal, and female breast [8]. Although research is still in its early stages in humans, researchers have inferred the mechanism by which alcohol promotes carcinogenesis from the studies of other animals [6-8]. Alcohol may promote carcinogenesis in its raw form by acting as a cellular membrane solvent, which increases permeability of other carcinogenic compounds present. However, more evidence links the consumption of alcohol to carcinogesis via the breakdown mechanism of ethanol. It is metabolized by alcohol dehydrogenases, cytochrome protein 4502E1 (CYP2E1), and bacterial alcohol dehydrogenases [6].

Tissue and bacterial alcohol dehydrogenases produce acetaldehyde and free radicals [6,7]. Both of these products induce mutations to the cell that lead to cancer, thus alcohol should be avoided in excess. Acetaldehyde is highly carcinogenic causing damage to DNA, which can initiate and progress tumor formation [6]. The free radical formation increases oxidative stress in the cellular environment which, as stated previously, is an inducer for the onset of cancer.

CYP2E1 also metabolizes ethanol to acetaldehyde and free radicals; however, the enzyme itself is an antagonist of normal cellular activity. This cytochrome protein also processes xenobiotics including procarcinogens [6,7]. There are a wide variety of sources of procarcinogens including components of cigarette smoke and foods; when they interact with CYP2E1, conversion may occur to an active carcinogen [9]. The interaction is dependent on many factors, so conversion is no absolute [7,9]. Along with the effects from acetaldehyde, free radicals, and CYP2E1, drinking elevated levels of alcohol enhances the deleterious effect of poor nutrition [7].

## Exercise overall

According to a recent study, involvement in exercise attributed to a 40% risk reduction in one's likelihood of developing cancer [10]. Exercise most greatly diminishes one's chances of developing colon and breast cancer, as well as prostate, endometrium, ovarian, and lung cancers [11]. Further research must be conducted to determine the effects that exercise has on other types of cancer.

## Obesity and central adiposity

The relationship between adipose tissue and cancer, especially colon, breast, endometrial, and ovarian, plays a big role in one's chances of developing cancer because many other causes of cancer stem from the presence of adipose tissue. Obesity accounts for 14% of cancers in men and 20% of cancers in women, which is why it is essential to maintain a healthy weight [9]. Extra adipose tissue creates an environment for many chemicals to circulate. For example, glucose, insulin, estrogens, testosterone, and adipokines are known to circulate in greater quantities in those with more adipose tissue [9,12]. High levels of these chemicals are all risk factors of cancer. Because extra adipose tissue is a host for these circulating chemicals, participating in exercise utilizing proper nutrition will lessen the risk of developing cancer by decreasing the amount of an individual's adipose tissue. Further, by creating this better proportion of circulating chemicals and adipose tissue, a better energy balance is created which is beneficial to preventing the onset of cancer [3].

Although all adipose tissue has been found to be a feeding ground for circulating chemicals, central adipose tissue could play the largest role in adipose tissues' relationship with the onset of cancer, because it is metabolically active. By devoting time to exercise and diet, one can decrease abdominal fat, therefore, reducing his or her chances of cancer onset [11].

## **Endogenous sexual hormones**

High levels of sexual hormones are attributed to the onset of certain types of cancer. This results from greater levels of adipose tissue, which leads to less hormone-binding globulin. This hormone binds to estrogen and testosterone, so without it, there are more circulating sexual hormones [12-14]. One can infer that having less adipose tissue, which can result from exercise and proper nutrition, allows for more hormone-binding globulin which causes the amount of circulating hormones to decrease. Additionally, exercise alone has been shown to increase the presence of sex hormone binding globulin [11,12]. Further supporting this claim, especially for the presence of estrogen, animal studies revealed that high volumes and intensities of physical activity changed sexual hormone levels.3 Therefore, the effects of exercise on sexual hormones has the greatest effects on reducing the chances of developing breast, endometrial, ovarian, and prostate cancer because these cancers are all effected greatly by the circulation of sexual hormones [15].

In terms of breast cancer specifically, exercise has shown to have different effects on premenopausal women versus postmenopausal. Exercise is important in premenopausal women because it helps to create less estrogen, delay menarches, and make menstrual cycles irregular if the levels of physical activity are high enough [8,11,12,16]. By delaying menarche and having irregular menstrual cycles, one will have less exposure to estrogens. Since exercise reduces body fat, there would be less fat-produced estrogens in one who exercises, which decreases one's chances as well [11].

Unlike premenopausal women, whose estrogen levels are significantly affected by ovaries, postmenopausal women's estrogen levels are most significantly affected by circulating estrogen in adipose tissue [13]. For this reason, it is extremely important that women participate in exercise and utilize proper nutrition, especially later in life when the presence of estrogen in adipose tissue will play the biggest role [17].

#### **Pulmonary function**

Studies agree that exercise can reduces the risk of lung cancer due to its effects on pulmonary function, although research is limited in this area. Exercise lowers the amount of foreign bodies in the lungs [12]. It also reduces the amount of time material is in the lungs and the interaction of that material [18]. Because one who smokes will have more carcinogenic material in the lungs, a smoker would more likely be effected by exercise. Further, smoking causes oxidative stress, which may be decreased through exercise and nutrition [18]. Because smoking plays a large role in determining the strength of the relationship, sometimes gauging strictly how exercise effects pulmonary function is difficult [11].

## Metabolic hormone levels and growth factors

Research has found that exercise will change metabolic hormone levels and growth factors in order to reduce the risk of cancer, especially the onset of colon, breast, endometrial, ovarian, prostate, and lung [12]. For example, exercise improves insulin sensitivity and lowers insulin, insulin-like growth factor, and glucose in the blood because of less body fat. Further, exercise increases muscle mass, allows insulin to bind to insulin-like growth factor binding protein, and transports glucose into the muscle, instead of circulating in the blood [11,12,19]. The decreased insulin and insulin-like growth factor also allows more normal cell division and inhibits apoptosis [11,18,20]. Lastly, exercise, along with a healthy diet, has also been shown to increase high-density lipoprotein cholesterol levels which reduce one's risk of cancer [18].

#### Immune system

Since a deficient immune system in general is associated with cancer, exercise's positive effect on the immune system plays a role in reducing the risk of most types of cancer. Exercise increases the amount of macrophages, cytokines, and lymphokines [11]. An increase in these cells can enhance antitumor immune defences because these cells work to eliminate tumor-forming carcinogens [3,11,15,18]. In addition to these effects on tumor development, both exercise and sound nutrition enhance antioxidant defence by increasing antioxidative enzymes that work to inhibit tumor growth and alter tumor development [3,11].

#### Limitations of the research

With any research, there are always limitations that affect the complete validity, standardization, and reliability of the findings. One of the biggest issue with exercise's and nutrition's relationship to the onset of cancer is that most of these studies were observational studies [4,21,22]. Although more difficult to perform, more studies need to be experimental if a causal relationship is to be found [12]. Even when experimental studies are done, however, there is still a chance that the subjects will not fully comply, and studies are only valid if the subjects actually do all of the directed exercise and consume the correct nutrients.

With observational studies where data collection formats are used such as questionnaires or surveys, issues with validity and standardization arise. For nutritional research, much of the data collection was based on questionnaires completed by the patients [4,21]. However, patients may not recall the exact timing of consumption of specific nutrients or remember consumption all together. Without knowing the length of time between consumption and diagnoses, it is difficult for researchers to determine the time in life certain foods are most productive in prevention.

Data collection formats raise issues with exercise research as well. When asked about previous exercise involvement, the subjects could have not been completely truthful or simply not remembered how often they exercised in the past; a phenomenon known as recall bias [6,12]. Even if the subjects were forthright, these questionnaires could have also lacked specification by not explaining what is meant by one hour of exercise. Most questionnaires did not specify the desired intensity, type, duration, or lifetime activity levels [20,22]. For example, to one subject, one hour of exercise could mean leisurely walking, while to another it means playing competitive tennis. These two different interpretations would have an impact on the intensity of their exercise and potentially explain why one subject reduced his or her risk and the other did not [8].

Additionally, some of the studies did not take their subjects' family history, lifestyle, and personalities into account [6,12,15]. As much as one would like to be able to control all potential causes of cancer, heredity, personality, and lifestyle factors do play a role in whether or not one will develop it. For example, maybe those who are more driven are less likely to get cancer and it has nothing to do with the exercise at all; simply, those who are more driven are more likely to exercise, which is why this correlation has been found.

#### Conclusion

Research findings clearly indicate a correlation between proper nutrition and exercise and a reduced risk of cancer. The additive effects of exercise and sound nutritional practices decrease the onset of cancer to a further extent. Although there are a large number of dietary constituents that could be tested with the multitude of human cancers, there is biological backing to the inferences drawn from the effects of specific nutrients.

#### References

- 1. Liu RH (2004) Potential synergy of phytochemicals in cancer prevention: mechanism of action. The Journal of Nutrition 134: 3479S-3485S.
- Birben E, Sahiner UM, Sackesen C, Erzurum S, Kalayci O (2012) Oxidative Stress and Antioxidant Defense. World Allergy Organ 5: 9-19.

- Rogers C, Colbert L, Greiner J, Perkins S, Hursting S (2008) Physical activity and cancer prevention, pathways and targets for intervention. Sports Med 38: 271-296.
- 4. Riboli E, Kaaks R (1997) The EPIC Project: Rationale and Study Design. Int J Epidemiol 29: S6-S14.
- Cross AJ, Leitqman MF, Gail MH, Hollenbeck AR, Schatzkin A, et al. (2007) A prospective study of read and processed meat intake in relation to cancer risk. PLoS Med 4: e325.
- Courneya K, Karvinen K, McNeely M, Campbell K, Brar S, et al. (2012) Predictors of adherence to supervised and unsupervised exercise in the alberta physical activity and breast cancer prevention trial. Journal of Physical Activity and Health 9: 857-866.
- 7. http://www.cdc.gov/cancer/dcpc/data/types.htm.
- Bernstein L, Henderson BE, Hanisch R, Sullivan-Halley J, Ross RK (1994) Physical exercise and reduced risk of breast cancer in young women. Journal of the National Cancer Institute 86: 1-5.
- 9. Eyre H, Kahn R, Robertson RM (2004) Preventing cancer, cardiovascular disease, and diabetes: A common agenda for the american cancer society, the american diabetes association, and the american heart association. American Cancer Society, American Diabetes Association, and the American Heart Association 54: 190-207.
- 10. Newton R, Galvao D (2008) Exercise in prevention and management of cancer. Current Treatment Options in Oncology 9: 135-146.
- 11. Friedenreich C, Orenstein M (2002) Physical activity and cancer prevention: Etiologic evidence and biological mechanisms. The Journal of Nutrition 132: 34565-34645.
- 12. Friedenreich CM, Neilson H, Lynch B (2010) State of the epidemiological evidence on physical activity and cancer prevention. European Journal of Cancer 2593-2604.

- Schmandt R, Iglesias DA, Co NN, Lu K (2011) Understanding obesity and endometrial cancer risk: Opportunities for prevention. American Journal of Obstetrics & Gynecology 518-525.
- 14. Young-McCaughan S (2012) Potential for prostate cancer prevention through physical activity. World Journal of Urology 30: 167-179.
- Norman A, Moradi T, Gridley G, Dosemeci M, Rydh B, et al. (2002) Occupational physical activity and risk for prostate cancer in a nationwide cohort study in Sweden. British Journal of Cancer 86: 70-75.
- Friedenreich C, Courneya K, Bryant H (2001) Influence of physical activity in different age and life periods on the risk of breast cancer. Epidemiology 12: 604-612.
- Monninkhof E, Elias S, Vlems F, Van der Tweel I, Schuit J, et al. (2007) Physical activity and breast cancer: A systematic review. Epidemiology 18: 137-157.
- Koutsokera A, Kiagia M, Saif M, Souliotis K, Syrigos K (2013) Nutrition habits, physical activity, and lung cancer: An authoritative review. Clinical Lung Cancer 14: 342-350.
- 19. Trojian T, Mody K, Chain P (2007) Exercise and colon cancer: Primary and secondary prevention. Current Sports Medicine Reports 6: 120-124.
- Wolin KY, Yan Y, Colditz GA, Lee I (2009) Physical activity and colon cancer prevention: A meta-analysis. British Journal of Cancer 100: 611-616.
- Key TJ, Schatzkin A, Willett WC, Allen NE, Spencer EA, et al. (2004) Diet, nutrition, and the prevention of cancer. Public Health Nutrition 7: 187-200.
- 22. Steindorf K (2013) The role of physical activity in primary cancer prevention. European Group for Research into Elderly and Physical Activity 10: 33-36.

Page 4 of 4