Cancer Prevention Through Employ of Appropriate Diet in Daily Schedule

Shukati Malik* and Sajid Khan

Department of Bioinformatics, Muhammad Ali Jinnah University Islamabad, Pakistan

Abstract

The aim of study is to evaluate the relationship between diet and cancer prevention. Cancer is one of the leading causes of death throughout the world. This study provides a summary of nutritional factors and their relation to risk of major cancers based on current data. We realize that supplemental nutrients may have different health effects than nutrients in food and that lifestyle behaviors such as smoking can modify risks. This research will provide new and better understanding of the complex physiological action of isolated supplements in health and disease.

Keywords: Diet and cancer hindrance; Fruits and vegetables; Relationship of diet schedule; Cancer

Introduction

Role of nutrition in cancer prevention

A diet rich in selenium and Omega-3 has a preventive role in prostate carcinoma. An excessive intake of food is one of the main factors of neoplastic risk and it is proved that obesity is a condition that predisposes the development of malignant neoplasm. Overweight is responsible for 14% of cancer deaths in men and 20% in women [11]. The major causative factors of cancer and their percentage has been explained (Table 1).

Fruits and citrus

One of the most important messages of modern nutrition research is that a diet rich in fruits protects against cancer. The greatest message is that this same diet protects against almost all other diseases, to, including cardiovascular disease and diabetes. There are many mechanisms by which fruits are protective, and an enormous body of research supports the recommendation for people to eat more fruits and vegetables. Overall, a high intake of fruits probably reduces the risks of cancers of the oral cavity, esophagus, stomach and colorectum [2].

<table>
<thead>
<tr>
<th>Tobacco</th>
<th>30%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet</td>
<td>35-60%</td>
</tr>
<tr>
<td>Air/Water Pollution</td>
<td>13%</td>
</tr>
<tr>
<td>Alcohol</td>
<td>17%</td>
</tr>
<tr>
<td>Radiation</td>
<td>3%</td>
</tr>
<tr>
<td>Medications</td>
<td>2%</td>
</tr>
</tbody>
</table>

Table 1: Some major causative factors of cancer and their percentage for causing cancer.

*Corresponding author: Shukati Malik and Sajid Khan, Department of Bioinformatics, Muhammad Ali Jinnah University Islamabad, Pakistan. E-mail: drsimalik@jinnah.edu.pk, sajiddilazakkhan@gmail.com

Received September 16, 2011; Accepted November 02, 2011; Published November 04, 2011


Copyright: © 2011 Malik S, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

ISSN:1948-5956 JCST, an open access journal

Special Issue 16 • 2011
At that time, however, the available literature was based largely on case-control studies, and subsequent prospective studies have not supported important protective effects for cancers of the lung and breast, and have suggested that the reduction in risk for colorectal cancer may be modest. These discordant results, which add to concerns about the potential for bias in case-control studies, also suggest the need for some caution regarding conclusions about intake of fruits and vegetables and the risks of oral, esophageal and stomach cancers, which have not been adequately examined in large prospective studies. Furthermore, none of the dietary studies of stomach cancer has controlled adequately for infection by H. pylori, which is a potential confounding variable [2].

Citrus fruit include Orange, Grapefruit, Lemon, and Mondon. Studies have suggested that photochemical compounds in citrus fruit block tumor growth by direct action on the cancerous cells, restricting their ability to reproduce. Such fruits consumption, whether in, the form of whole fruit or juice, supplies the body with an incomparable polyphenols known to affect cancer cell growth in vitro. Very little is known about the effects of tea consumption on cancer survivor. Moderate amounts of green or black tea can be considered safe and helpful in preventing the cancer [14].

Berries

Most berries are an exceptionally abundant source of several classes of polyphenols that possess anticancer potential ellagic acid, anthocyanidins, and proanthocyanidins. Eating cranberries should be preferred over drinking cranberry juice [12].

Macronutrients

Carbohydrates, protein, and fat each contribute energy to the diet, so excess energy intake resulting from overconsumption of any or a combination of these macronutrients may influence cancer risk. However, the question of whether individual macronutrients increase risk independent of energy balance remains controversial [15].

For example, in intervention studies of low-fat diets under is caloric conditions, calories from fat are replaced with calories from carbohydrate and/or protein. Other nutrients are likely to change as well. As a result, differentiating the effect of reduced fat intake from these other changes is difficult. Likewise, simply adding fat to the diet cannot be separated from adding energy (or the consequent weight gain). Epidemiologic studies of macronutrients must therefore carefully consider total energy and other energy-bearing nutrients in the analysis [15-17].

Fat

The results of studies of fat consumption in relation to cancer have been inconsistent, particularly across study designs. Much of the interest regarding nutrition and cancer originated from ecologic studies, in which countries with high per capita fat intake were shown to have higher rates of cancers of the breast [18,19], colon, and prostate [20,21] than countries with lower fat intakes. As important confounders were not measured and controlled for in these analyses, dietary fat may also have been merely a marker for a true causal factor (e.g. reproductive factors). Animal studies also contributed to the hypothesis that fat caused cancer but fat intake was likely to be a surrogate of energy intake [22]. Fat intake has been hypothesized to increase the risk of breast and prostate cancer by modulating sex hormone levels, and to increase colon cancer risk by stimulating mutagenic secondary bile acid secretion. Early dietary guidelines, based on these data, emphasized fat reduction for cancer prevention [23].

Carbohydrates

Coincident with the strong emphasis on lowering dietary fat over the past several decades, grain and sugar consumption in the US increased markedly. Carbohydrates are heterogeneous and probably have varying effects on health and disease [24].

Dietary carbohydrates include starches (e.g. bread, pasta, other grains), non-starch polysaccharides (the major component of dietary fiber), and sugars. Carbohydrates with a high glycemic index [25] are associated with higher postprandial blood glucose and insulin [26,27], and higher fasting insulin levels in insulin-resistant states [25], and are thus hypothesized to increase cancer risk. However, epidemiologic studies currently provide limited support for a direct role of diets high in glycemic load (which takes the total carbohydrate intake into account) in cancer development.

Some studies of colon and breast cancer [26,27] did not find an association between diets high in glycemic load or sugar and cancer, while others did [28-30]. The inconsistencies may result from difficulties in measuring the glycemic potential of diet, given the

importance of meal composition, for example. However, glycemic load measured from FFQs has strongly predicted the risk of coronary heart disease [31] and type II diabetes [32]. Glycemic load may increase the risk particularly among susceptible subgroups; one study observed increased risk of breast cancer only among those with elevated BMI [33,34]. Pancreatic cancer risk was increased by 53% with a high glycemic diet in a study of women, and by 170% among those who were sedentary and overweight. More work is needed, as most studies of carbohydrates and cancer risk have not considered carbohydrate quality. Nonetheless, the existing data suggest that abnormal glucose and insulin metabolism is important in carcinogenesis, especially in obese, sedentary individuals [35].

Meat

Evidence for a role of meat consumption in increasing cancer risk, especially of the colon, rectum, and prostate, has been fairly consistent over time and across study designs. Countries with high per capita meat consumption were shown to have higher incidence of colon cancer than those with low meat consumption. Two meta-analyses of meat and colorectal cancer risk were recently published, one including 13 prospective studies [36], and the other including results from 34 case–control studies and 14 prospective cohort studies. The former reported a significant 12–17% increase in risk associated with each daily 100 g increment of all meat or red meat intake (slightly more than 3 oz), and a 49% increased risk for each 25 g increment of processed meats (about one slice) [37]. In the other meta-analysis, the investigators reported a 24% increase in risk associated with each daily 120 g increment of red meat intake, and a 36% increase in risk for each 30 g increment of processed meat [38,39].

Summary

The evidence for a role of diet in cancer prevention is continually evolving as new studies accumulate. This study provides a summary of nutritional factors and their relation to risk of major cancers based on current data. The factors listed would undoubtedly vary if summarized by other authors, but the evolving and expanding is does demonstrate substantial progress since Doll and Peto’s [40,41] report. For example, leading hypotheses even 10 years ago centered on total calories, dietary fat, specific factors in fruits and vegetables (including betacarotene and dietary fiber), and vitamins A, E, and C [42]. These hypotheses have been tested, extended, and clarified, and some have been refuted. A role for positive energy balance and obesity in carcinogenesis, especially in the insulin resistance syndrome, is generally accepted. Nonetheless, the existing data suggest that abnormal glucose and insulin metabolism is important in carcinogenesis, especially in obese, sedentary individuals [35].

Acknowledgment

We would like to thank Ms Z. Khalid & S. Zahra for helping in the proof reading of the paper.

References


