

A Short Diet, Nutrition-An Approach to the Prevention of Cancer

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Citation: Nasir A (2020) A Short Diet, Nutrition-An Approach to the Prevention of Cancer. *J Food Nutr Health*, Volume 1:1. 101. DOI: <https://doi.org/10.47275/2692-5222-101>.

Received: February 04, 2020; Accepted: March 27, 2020; Published: April 02, 2020

Introduction

Diet and lifestyle conditions in Western states were believed to contribute to around 30 percent of cancers [1]. Adjusting diet second only to the preventive cause of tobacco cancer. The dietary contribution of the emerging regions to the risk of cancer was found lower, maybe about 20 percent [2]. Consequently, uncovering the impact of diet on cancer risk is of considerable significance for public health, but work to date has identified a few clear effects and left irritatingly broad discrepancies. Most of the influential explanations for nutritional impact on cancer risk were developed from analyzing the correlations among lifestyle habits and cancer levels in various communities across the globe. It was reported that industrialized Western nations had foods that are high in meat and dairy, sugar and fat, and leads to high levels of different cancers. Many experiments showed that cancer levels frequently shift among societies that move from one region to the next, and shift in nations over the period. For reference, Japanese populations previously low colorectal cancer levels have risen both in terms of relocation to the United States and, very recently, in Japan's [3].

Western diet

As mentioned, the findings of human diets have not confirmed any of the key theories that were extracted from these ecological findings. Global differences in diet and cancer levels, moreover, tend to indicate that diet is a significant risk factor for several forms of cancer and that disease can be significantly avoided by dietary changes. Prevalence levels reported for several basic conditions globally in the past [4]. Lung cancer is the globe's complex cancer and also the most common cancer for men across both developed and developing countries, while breast cancer is common among females. The archetypal Western cancers are colorectal, breast, and prostate cancers. Westernization involves numerous improvements in lifestyle and diet, involving higher meat consumption, milk products, sugar, and other processed carbohydrates, and decreased intakes of comparatively unrefined staple starchy foods. Like nutrients, modern diets are characterized by sufficient or extreme energy intake, along with high protein and fat intakes, while in nation's with middle-income micronutrient deficiencies are far more severe. The research on the nutritional reasons of typical Modern cancers has concentrated primarily on increased ingestion of macronutrients, while the hunt for the nutritional reasons of emerging countries' modern

cancers has centered on micronutrient deficiency intake. Many studies have been conducted over the past 50 years which have explored the relationship between specific diets and the likelihood of developing the disease. Almost all previous research was performed using a case study approach in which individuals with cancer are questioned what they'd have to eat.

The case studies are helpful for potential dietary consequences, but cannot be relied on to create reasonable dietary relationships, because they are susceptible to both remember and preference biases: cancer patients can remember certain diet separately from healthy individuals, and healthy controls are rarely completely substituted of the bottom group and may describe a comparatively poor diet [5,6]. Dietary managements are assessed at induction and individuals are followed up for cancer occurrence, and levels are then matched at baseline among different people with different intakes. The architecture removes the memory and classification limitations that are vulnerable to case studies, but some variables like measurement of error need to be addressed when evaluating the findings of potential diet and research. A further drawback in certain cohort trails would be specific dietary consumption is assessed at a phase throughout adulthood that may not be a close enough time to bond to cancer growth over several decades. Broad retrospective research will assess the correlation among recorded diets or hazards of cancer inside the examined samples. Experimental failure and uncertainty are the key factors that must be addressed when assessing findings from these studies. A comparatively small and easy dietary application is commonly used in epidemiological investigations. Efficacy of the tests has been thoroughly studied, and although it is evident that existing evaluation techniques are fairly reliable and can identify some dietary connections with disease prognosis, it is also important that the sampling error is often highly adequate to uncertain and certain possibly significant dietary interactions with cancer risk. Another issue is the clear similarities among various foods and components, making it difficult to assign risk interactions to specific dietary factors, particularly when the calculation of accuracy is small. Body mass index isn't just a diet per se, but is calculated by the amount of calorie gain and energy investment; consequently, body mass index acts as a marker of chronic energy equilibrium, and can be assessed more consistently and correctly in epidemiological investigations than either energy eating or energy investment. Drinking is not often incorporated in the word diet, but



in many societies, alcoholic beverages add a substantial strength supply and certain nutrients. While evaluation of alcohol intake often suffers from failings to report, they typically give an order for people much more accurately than measures of other foods and nutrients intake. Randomized observational experiments remove both the prejudices and the uncertainty that may influence empirical research, and thus the findings may be explained with trust in consideration of purpose and impact. Furthermore, in the context of diet and disease, research is constrained by the difficulties of randomizing at the food stage, and by the limitations that only a small proportion of nutritional aspects can be evaluated in each study, generally for a short span of time. The outcomes of the few big experiments that have been conducted are significant, but those studies do not demonstrate an impact it appears likely that an impact may have been shown at a specific dosage, at a specific time in life, or if the length of the study was larger. Some other factor that should be addressed when analyzing the findings of randomized clinical trials is the probability that the impact of a dietary variable on the risk of cancer may vary based on the features of the community researched; for instance, the consequences of a multi-nutrient replacement in a community with a low dietary intake of micronutrients may be more pronounced than in the community. Randomized experiments are ideally adapted in the area of diet and cancer to investigate theories for the beneficial impacts of different micronutrients. The first IARC Handbook of Cancer precaution offers a comprehensive analysis of such theories and the nature and understanding of these experiments [7]. It was estimated that the oral cavity, pharynx, and esophagus cancers account for 867,000 cases and 582,000 deaths in 2000 [8]. The overall rate of these cancers varies widely among communities; for example, esophageal cancer is more than a hundred times more common in parts of China, Central Asia, and South Africa than in the greatest parts of North America, West Africa, and Europe [9]. The key risk conditions in developing nations are liquor and smoking, and these specific external factors cause up to 3/4 percent of all these cancers. The process of alcohol's impact on such cancers is unclear, but may have direct consequences on the epithelium [10]. Fatty foods and or malnutrition are known risk indicators, especially for esophageal adenocarcinoma. In developed nations, about 60 percent of oral, pharynx, and esophageal cancers are probably related to micronutrient insufficiencies associated with a limited diet low in fruit, vegetables, and animal products [11-14].

In communities with Western cancer incidence trends, the nutritional aspect for which the information was regarded most credible, and for which the quantitative influence on overall cancer rates is most valuable, is overweight / obesity. Overweight/obesity is credibly correlated with the risk of oesophageal, colorectal, breast, endometrial, and kidney cancers. Specifically, the increased incidence of these cancers progressively rises with increased adiposity and is not restricted to clinical obesity. The rapid increase in endogenous estrogen levels of many postmenopausal women due to excess body fat likely explains the increased threats of postmenopausal breast and endometrial cancer. The pathways for other cancers are less evident, but hyperinsulinemia has been suggested to raise the risk of colon cancer [15]. The WHO / IARC working group on overweight management and physical exercise reported that excess body weight accounts for about 39 percent of endometrial, 25 percent of prostate, 11 percent of colon, 9 percent of postmenopausal breast cancer and 5 percent of overall cancer rates in countries with high levels of cancer linked to overweight [16,17]. In developing countries, the rapid increase in overweight / obesity means that cancers due to overweight / obesity will become increasingly important worldwide. Acknowledging that

overweight/obesity, a consequence of over energy consumption, may lead to physical activity from both excessive consumption of calories from food and low energy investment; the comparative significance of these two causes that differ between individuals and communities. The probability that the content of the diet affects the likelihood of fat accumulation and thus, consequently, the risk of cancer, has been given significant interest. Whereas the amount of dietary fat energy was hypothesized to be an essential factor of body fat and the subject was problematic, a significant impact of dietary fat was not confirmed in randomized trials lasting a year or longer, and populations eating low fat and or high carbohydrate diets can establish high levels of obesity [18]. Although these levels would be lower in communities in some developed countries where virally associated cancers are more significant, in developing countries, the rapid increase in overweight and obesity means that cancers due to these factors will become increasingly important worldwide. Though, the information for the beneficial effect of fruit and vegetables is mainly obtained from case study research and there is still little data from planned research. The corresponding functions of the different micronutrients are not yet clear, but riboflavin, folate, vitamin C, and zinc deficiencies may all be significant [19]. There is also strong evidence that consuming extremely high temperatures of beverages and foods raises the risk of these cancers.

References

1. Doll R, Peto R (1981) The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. *J Nat Cancer Inst* 66:1192-1308.<https://doi.org/10.1093/jnci/66.6.1192>
2. Greenwald P, Clifford CK, Milner JA (2001) Diet and cancer prevention. *Euro J Cancer* 37:948-965.[https://doi.org/10.1016/S0959-8049\(01\)00070-3](https://doi.org/10.1016/S0959-8049(01)00070-3)
3. Stewart BW, Kleihues P (2003) World cancer report. International Agency for Research on Cancer, IARC Press, United Kingdom.
4. Ferlay J, Colombet M, Soerjomataram I, Mathers C, Parkin DM, et al. (2019) Estimating the global cancer incidence and mortality in 2018: GLOBOCAN sources and methods. *Int J Cancer* 144:1941-1953.<https://doi.org/10.1002/ijc.31937>
5. Miller AB, Kelly A, Choi NW, Matthews V, Morgan RW, et al. (1978) A study of diet and breast cancer. *Am J Epidemiol* 107:499-509.<https://doi.org/10.1093/oxfordjournals.aje.a112569>
6. Margetts BM, Nelson M (Eds) (1998) Design concepts in nutritional epidemiology. Oxford University Press, New York, United States.
7. Morse MA, Stoner GD (1993) Cancer chemoprevention: principles and prospects. *Carcinogenesis* 14:1737-1746.<https://doi.org/10.1093/carcin/14.9.1737>
8. Mignogna MD, Fedele S, Russo LL (2004) The World Cancer Report and the burden of oral cancer. *Euro J Cancer Prev* 13:139-142.<https://doi.org/10.1097/00008469-200404000-00008>
9. Ferlay J (2004) Cancer incidence, mortality and prevalence worldwide. GLOBOCAN2002.
10. Smoke T, Smoking I (2004) IARC monographs on the evaluation of carcinogenic risks to humans. IARC 2004:1-452.
11. Chen H, Ward MH, Graubard BI, Heineman EF, Markin RM, et al. (2002) Dietary patterns and adenocarcinoma of the esophagus and distal stomach. *Am J Clin Nutri* 75:137-144.<https://doi.org/10.1093/ajcn/75.1.137>
12. Cheng KK, Sharp L, McKinney PA, Logan RF, Chilvers CE, et al. (2000) A case-control study of oesophageal adenocarcinoma in women: a preventable disease. *Br J Cancer* 83:127-132.<https://doi.org/10.1054/bjoc.2000.1121>
13. Giacosa A, Franceschi S, La CV, Favero A, Andreatta R (1999) Energy intake, overweight, physical exercise and colorectal cancer risk. *Eur J Cancer Prev Org* 8:S53-S60.<https://doi.org/10.1097/00008469-199912001-00009>
14. Kinlen LJ (1991) Cancer: causes, occurrence and control. *Br J Cancer* 64:977-978.
15. Colangelo LA, Gapstur SM, Gann PH, Dyer AR, Liu K (2002) Colorectal cancer mortality and factors related to the insulin resistance syndrome. *Cancer Epidem Biomar Prev* 11:385-391.



16. Giovannucci E (1995) Insulin and colon cancer. *Cancer CausCon* 6:164-179.
17. Ceschi M, Gutzwiller F, Moch H, Eichholzer M, Probst-Hensch NM (2007) Epidemiology and pathophysiology of obesity as cause of cancer. *Swiss medical weekly* 137:50-56. <https://doi.org/10.1007/BF00052777>
18. Willett WC (1998) Is dietary fat a major determinant of body fat?. *Am J Clin Nutr* 67: 556S-562S. <https://doi.org/10.1093/ajcn/67.3.556S>
19. Boffetta P, Garcia-Gómez M, Pompe-Kirn V, Zaridze D, Bellander T, et al. (1998) Cancer occurrence among European mercury miners. *Cancer Caus Cont* 9:591-599. <https://doi.org/10.1023/A:1008849208686>