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## RESEARCH COMMUNICATION

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# A Low Fat Diet Rich in Fruits and Vegetables May Reduce the Risk of Developing Prostate Cancer

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### Abstract

**Background:** Diet has been implicated in prostate cancer risk and there is evidence of risk reduction with a healthy diet. The objective of this population-based case control study was to examine whether a low fat diet rich in fruits and vegetables can reduce the risk of developing prostate cancer in Mumbai, India. **Methods:** Included in this study were microscopically proved cases of prostate cancer diagnosed during 1998 to 2000 and registered by Bombay Population Based Cancer Registry (n=594). The controls were healthy men belonging to the resident general population of Mumbai, India. Two controls for each case matched by age and place of residence were selected as the comparison group. Data on oil/fat consumption, fruits and vegetable consumption and other probable confounding factors were obtained by structured face-to-face interview. After exclusions, 390 cases and 780 controls were available for final analysis and confounding was controlled by multiple logistic regression. **Results:** 58.7% of the control group consumed more than 3 kg of fruits and vegetables per week compared to 52.1% of the case group. Controlling for age and probable confounding factors, a statistically significant protective effect for prostate cancer was observed for those who consumed fruits and vegetables 2 to 3 kg (OR 0.5, 95% CI 0.3-0.8) and more than 3 kg (OR 0.4, 95% CI 0.3-0.6) per week compared to those who consumed less than 2 kg per week. The linear trend for the protective effect was highly significant with increase in the consumption of fruits and vegetables ( $p = 0.001$ ). Even though not statistically significant, oil/fat consumption showed an elevated risk (OR 1.7, 95% CI 0.9-3.3) for those who consumed more than 2kg of oil/fat per month compared to those who consumed less than 1kg. **Conclusion:** The findings from this study support the hypothesis that a low fat diet rich in fruits and vegetables may reduce the risk of prostate cancer.

**Key Words:** Fruits and vegetables - oil/fat - prostate cancer - case control study

*Asian Pacific J Cancer Prev*, 6, 490-496

### Introduction

Prostate cancer has become a major public health burden worldwide with an estimated number of 679,000 new cases in the year 2002. This represents 11.7% of all new cancer cases in men (19.03% in developed countries and 5.3% in developing countries) (Ferlay et al. 2004).

Diet has been implicated in prostate cancer risk. Studies suggest that men can reduce their risk of prostate cancer by a healthy diet. People in countries such as China and Japan are far less likely than Westerners to develop prostate cancer. Notably, when people migrate to the US, their rates of prostate cancer rise greatly and, since their genetic make-up is the same, there appears to be something about living in America that increases these men's chances of developing prostate cancer. Diet is the number one suspect.

An interesting observation is that although the incidence of latent (occult, histologically evident) prostate cancer is similar throughout the world, clinical prostate cancer varies

from country to country by as much as 20-fold (Wynder et al. 1971). Previous ecologic studies have demonstrated a direct relationship between a country's prostate cancer-specific mortality rate and average total calories from fat consumed by the country's population (Armstrong and Doll 1975, Rose and Connolly 1992). Studies of immigrants from Japan have demonstrated that native Japanese have the lowest risk of clinical prostate cancer, first generation Japanese-Americans have an intermediate risk, and subsequent generations have a risk comparable to the U.S. population (Haenszel and Kurihara 1968, Shimizu et al. 1991). Animal models of explanted human prostate cancer have demonstrated decreased tumor growth rates in animals fed a low-fat diet (Wang et al. 1995, Connolly et al. 1997).

Increased dietary intake of fruits and vegetables has been associated with a reduced risk of prostate cancer in some studies. Intake of legumes, yellow-orange, and cruciferous vegetables were associated with a lower risk of prostate cancer (Kolonel et al. 2000). A significant difference between

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Asian and Western diets is the average amount of soy protein consumption (Yip et al. 1999). There is current interest in the possibility that the low risk of prostate cancer in certain Asian populations may result from their high intake of soy products (Miller et al. 1993). Soya beans are widely known for their possible anti-cancer potential, and the latest discoveries are encouraging. It has been found that the beans, which contain compounds called isoflavonoids, can actually inhibit prostate cancer cell growth in the laboratory. Soya protein can be found in many foods such as tofu, soya milk and yoghurt. The Eastern diet is based around soya proteins as an alternative to meat. In one study, Japanese men in Hawaii who consumed tofu approximately once per day, were 65 per cent less likely to develop prostate cancer in comparison with men eating tofu less than once in a week (Severson et al. 1989). In another study, Seventh-day Adventist men in California who consumed soy milk more than once daily were 70% less likely to develop prostate cancer as men who did not consume soy milk (Jacobsen et al. 1998). The pronounced protective effects of soy consumption in these studies are striking. Further work needs to be done to understand its effects on prostate cancer growth and role as dietary inhibitor of prostate cancer development and growth.

Micronutrients in the diet have also been implicated in the pathogenesis of prostate cancer (Platz et al. 1999, Nomura et al. 1997, Hsing et al. 1990a). Many studies showed that men with the highest level of selenium had one-third the risk of developing prostate cancer compared with men with the lowest selenium levels (Heinonen et al. 1998, Clark et al. 1997). One of the richest sources of selenium is brazil nuts. Selenium is also found in sunflower seeds, whole wheat bread, avocados and lentils and has been shown to reduce the incidence of prostate cancer. The association between prostate cancer and baseline vitamin E and selenium was evaluated in the trial-based cohort of the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study (ATBC). There were no significant associations between baseline serum alpha-tocopherol, dietary vitamin E, or selenium and prostate cancer overall. The associations between prostate cancer and vitamin E and some of the baseline dietary tocopherols differed significantly by alpha-tocopherol intervention status, with the suggestion of a protective effect for total vitamin E among those who received the alpha-tocopherol intervention (Hartman et al. 1998).

A variety of carotenoids, including lycopene, inhibit prostate cancer cells in vitro (Yip et al. 1999). A higher intake of lycopenes, the agent in tomatoes and beets that gives them their red color, has been shown to decrease risk of prostate cancer (Gann et al. 1999). In a large study of food intake and risk of prostate cancer, Giovannucci and colleagues (1995) demonstrated an inverse relationship between consumption of lycopenes and the risk of prostate cancer. The major dietary sources of lycopenes are cooked tomatoes, tomato juice, and paste. The role of vitamin A in prostate cancer growth is less established (Reichman et al. 1990).

Several randomized trials are ongoing evaluating the role of nutrition in preventing prostate cancer progression, but these results will not be available for several years.

A variety of fruits and vegetables are consumed by Indians at varying levels. It is difficult to get the information of specific fruits and vegetables items consumed by the study subjects in India. So we have combined fruits and vegetables and measurements were made on an average basis as how many kilograms of fruits and vegetables were consumed by the subjects in a week. The measurements for oil/fat consumption by the study subjects were also made in a similar way. The aim of the present study was to examine whether a low fat diet rich in fruits and vegetables can reduce the risk of prostate cancer.

## **Materials and Methods**

This study was planned and conducted as a matched case-control study. The cases were all prostate cancer patients registered by the Bombay Population Based Cancer Registry during the period, 1st January 1998 to 31st December 2000. Cases who had a microscopic proof of diagnosis were included in the study. Two controls were elected for each case to ensure enough power for the study. The controls were elected from the neighborhood of each case aiming at a maximum age difference of +5 years between the case-control pair/triplet.

During the period 1998 to 2000, a total of 766 prostate cancer cases were registered by the Bombay PBCR. Out of these, 172 (22.5%) cases that were not having any microscopic proof of diagnosis were excluded from this study. An attempt had been made to interview all the 594 (77.5%) cases with microscopic proof of diagnosis and their respective age matched controls.

Keeping the objective of this study as the guideline, a questionnaire was prepared for data collection. The questionnaire consisted of the following sections,

1. Identification particulars
2. Socio-demographic parameters
3. Oil/Fat consumption
4. Fruits and vegetable consumption
5. General dietary patterns
6. History of vasectomy
7. Tobacco and alcohol habits

The questions were constructed by an expert committee that consisted of clinicians and epidemiologists.

One trained male social investigator was appointed to collect the exposure history of the cases and controls. An appeal letter signed by the principal investigator of our organization was provided in order for a permission to interview the cases and controls. The interview has been carried out for all eligible cases and respective controls from 1st November, 1999 to 30th October 2001. After interviewing a case, all efforts were made to interview simultaneously two controls. For those cases having a single control or no controls, a second and third visit was made within a week time in order to find out respective controls.

To collect the exposure history of cases who already died before an interview has been made by interviewing the nearest relative of the patient, that is, either wife or son or male sibling. Over 95% of the proxy respondents were either wife or son or male sibling and 5% were others.

Although all efforts have been made to interview all the 594 microscopically proved cases and respective age matched controls, two controls for each case, only 390 cases (65.7%) and 780 controls were available to be included in the study for final analysis. The reasons for excluding the remaining 204 cases (34.3%) were due to migration, door closed, not willing to give personal details, too old / died and proxy respondents were not available or not cooperative to complete the interview, no eligible controls were available in the neighborhoods, controls were not willing to give personal details etc. Among the 390 cases included in the final analysis, 142 cases were estimated to have the time of death before the time of interview. Therefore the information was received by proxy respondents. In another 14 cases the interview was known to be based on proxy respondents mainly because of advanced disease and severe condition of the patient. The information for a total of 156 (40.0%) cases was therefore known to be collected by interviewing proxy respondents. For the remaining 234 (60.0%) cases, information was reported by self. For all the 780 controls included in the final analysis, information was reported by self. Masking of the investigator for data collection could not be ensured although the objectives/hypothesis of the study was not made known to him.

For some of the cases, it was not possible to find the proper age matched controls residing in the neighborhood of the respective cases due to less number of old age men in the general population of Mumbai. More than 60% of the age matched case-control triplets included in the study for final analysis had an age difference of more than +5 years between the case-control pairs or triplets, mainly because the neighborhood controls were younger than the cases. Hence a failure in age matching occurred.

The data collected by the social investigator was compiled and quality checks were carried out. Due to failure in age matching, unconditional logistic regression method was used for risk estimates. The details of the methods were taken from Breslow and Day (1980).

The multiple logistic regression method was employed to estimate the adjusted odds ratios for fruits and vegetables and oil/fat consumption with 95% CI. Since the etiology of prostate cancer is largely unknown, the adjusted odds ratios for fruits and vegetables and oil/fat consumption were obtained by adjusting with age and other probable confounding factors under study. The analysis was conducted using the Stata statistical software (version 7.0).

## Results

The distribution of the cases and controls by age-group, socio demographic, dietary, and lifestyle characteristics are presented in Table 1. There was a significant difference of

6.8 years between the mean age of the cases (71.2) and controls (64.4) and so the controls were younger than the cases.

The adjusted odds ratios for fruits and vegetables and oil/fat consumption were estimated by adjusting with age and all other probable confounding factors.

58.7% of the control group consumed more than 3 kg of fruits and vegetables in a week compared to 52.1% of the case group. 31.7% of the control group consumed 2 to 3 kg of fruits and vegetables in a week compared to 26.4% of the case group. Controlling for age and probable confounding factors, a statistically significant protective effect was observed for those who consumed fruits and vegetables 2 to 3 kg (OR 0.5, 95% CI 0.3-0.8) and more than 3 kg (OR 0.4, 95% CI 0.3-0.6) in a week compared to those who consumed

**Table 1. Distribution of Cases (n=390) and Controls (n=780) by Age Group, Socio Demographic, Dietary, and Lifestyle Characteristics**

Characteristic	Cases N (%)	Controls N (%)
<b>Age group</b>		
<55	16 (4.1)	58 (7.4)
55-64	63 (16.2)	305 (39.1)
65-74	163 (41.8)	338 (43.3)
75-84	108 (27.7)	71 (9.1)
85+	40 (10.3)	8 (1.0)
Mean	71.2	64.4
<b>Age at marriage</b>		
<=19	82 (21.0)	200 (25.6)
20-24	244 (62.6)	541 (69.4)
25+	64 (16.4)	39 (5.0)
Mean	21.2	20.4
<b>Vasectomy</b>		
No	332 (85.1)	702 (90.0)
Yes	58 (14.9)	78 (10.0)
<b>Fish (kg per week)</b>		
No	154 (39.5)	248 (31.8)
<1	100 (25.6)	239 (30.6)
1+	136 (34.9)	293 (37.6)
<b>Meat (kg per week)</b>		
No	109 (27.9)	183 (23.5)
<1	119 (30.5)	274 (35.1)
1+	162 (41.5)	323 (41.4)
<b>Coffee (cups per week)</b>		
<2	260 (66.7)	473 (60.6)
2+	130 (33.3)	307 (39.4)
<b>Tea (cups per week)</b>		
1-2	26 (6.7)	32 (4.1)
3-6	240 (61.5)	549 (70.4)
7+	124 (31.8)	199 (25.5)
<b>Tobacco smoking</b>		
No	94 (24.1)	148 (19.0)
Yes	296 (75.9)	632 (81.0)
<b>Tobacco chewing</b>		
No	339 (86.9)	669 (85.8)
Yes	51 (13.1)	111 (14.2)
<b>Alcohol drinking</b>		
No	113 (29.0)	111 (14.2)
Yes	277 (71.0)	669 (85.8)

less than 2 kg in a week. The linear trend in the protective effect was highly significant with increase in the consumption of fruits and vegetables ( $p$  for trend = 0.001). Even though not statistically significant, oil/fat consumption showed an elevated risk (OR 1.7, 95%CI 0.9-3.3) for those who consumed more than 2kg of oil/fat in a month compared to those who consumed less than 1kg oil/fat per month.

## Discussion

Increased dietary intake of fruits and vegetables has been associated with a reduced risk of prostate cancer in some studies (Mills et al. 1989, Gann et al. 1999, Kolenel et al. 2000, Jian et al. 2005). In the present study, consumption of more quantities of fruits and vegetables (2 to 3 kg and more than 3 kg per week) showed a protective effect for prostate cancer compared to those who consumed less than 2 kg per week. The present study results were consistent with many of the earlier studies. In a recent case-control study conducted in Hangzhou, southeast China during 2001–2002, the prostate cancer risk was declined with increasing consumption of lycopene, alpha-carotene, beta-carotene, beta-cryptoxanthin, lutein and zeaxanthin, and intake of tomatoes, pumpkin, spinach, watermelon and citrus fruits were also inversely associated with the prostate cancer risk (Jian et al. 2005). The corresponding dose-response relationships were also significant, suggesting that vegetables and fruits rich in lycopene and other carotenoids may be protective against prostate cancer (Jian et al. 2005). In another study, it has been shown that a higher intake of lycopenes, the agent in tomatoes and beets that gives them their red color, has been shown to decrease risk of prostate cancer (Gann et al. 1999). In a large study of food intake and risk of prostate cancer, Giovannucci and colleagues (1995) demonstrated an inverse relationship between consumption of lycopenes and the risk of prostate cancer. The major dietary sources of lycopenes are cooked tomatoes, tomato juice, and paste. In a multicentric, multi-ethnic population based case control study, it was found that intake of legumes, yellow-orange, and cruciferous vegetables was associated with a lower risk of prostate cancer (Kolenel et al. 2000). Another prospective study of Seventh-day Adventist men (Mills et al. 1989), it was found that

consumption dietary items such as beans, lentils, peas, and tomatoes were significantly associated with lower risk for prostate cancer. A variety of carotenoids, including lycopene, inhibit prostate cancer cells in vitro (Yip et al. 1999).

In a case-control study in Japan (Ohno et al. 1988), fruit consumption was associated with a moderate increase in prostate cancer risk. In another case control study in Japan, consumption of fruits, all vegetables, green-yellow vegetables, and tomatoes showed no association, but the study supported the hypothesis that the traditional Japanese diet, which is rich in soybean products and fish, might be protective against prostate cancer (Sonoda et al. 2004). A case-control study from Canada (Jain et al. 1999) reported a statistically significant positive association between prostate cancer and the intake of total fruit, citrus fruit, and fruit other than citrus. Fruits and fruit juices as a single category were significantly positively associated with prostate cancer in another recent Canadian case-control study (Villeneuve et al. 1999). Fresh fruit showed no association with prostate cancer in a case-control study in Italy (Talamini et al. 1986), and the mean weekly consumption of total fruits was similar for cases and controls in a study conducted in China (Lee et al. 1999). Among cohort investigations, no clear association with total fruit intake was seen in five studies (Snowdon et al. 1984, Shibata et al. 1992, Hsing et al. 1990b, Mills et al. 1989, Giovannucci et al. 1995). In a subsequent report on one of these cohorts (Giovannucci et al. 1995), a protective effect against advanced prostate cancer was seen for fruits; this finding was accounted for by fructose intake (Giovannucci et al. 1998). Two other cohorts (Severson et al. 1989, Schuurman et al. 1998) found an increased risk associated with total fruit intake; the result was statistically significant in one of these studies (Schuurman et al. 1998). One cohort study (Mills et al. 1989) found a weak inverse association for dried fruits (raisins, dates, and others); another (Severson et al. 1989) found a statistically significant increase in risk for citrus fruits. The findings from prospective cohort studies are also inconclusive. Two studies found no association for total vegetables (Snodon et al. 1984, Shibata et al. 1992), and one study found no association for cruciferous vegetables in particular (Hsing et al. 1990a). A study in Japan reported a protective effect of green-yellow vegetables in men, 75 years of age, whereas a study of

**Table 2. Univariate and Adjusted Odds Ratios for Fruits and Vegetables, Oil/Fat by Unconditional Logistic Regression Method for (n=390) Cases and (n=780) Controls**

Characteristic	Cases N (%)	Controls N (%)	Univariate OR (95% CI)	Adjusted* OR (95% CI)	p trend
Fruits and vegetables (kg per week)					
<2	84 (21.5)	75 (9.6)	1.0	1.0	
2–3	103 (26.4)	247 (31.7)	0.4 (0.3-0.5)	0.5 (0.3-0.8)	
>3	203 (52.1)	458 (58.7)	0.4 (0.3-0.6)	0.4 (0.3-0.6)	0.001
Oil/Fat (kg per month)					
<1	69 (17.7)	138 (17.7)	1.0	1.0	
1–2	291 (74.6)	617 (79.1)	0.9 (0.7-1.3)	0.9 (0.6-1.3)	
>2	30 (7.7)	25 (3.2)	2.4 (1.3-4.4)	1.7 (0.9-3.3)	0.52

\* the adjusted odds ratios were obtained by adjusting with age and other probable confounding characteristics under study

Japanese men in Hawaii (Severson et al. 1989) reported an increased risk in men with higher seaweed consumption. Two cohort studies found an inverse association with the consumption of tomatoes but not of total vegetables or of other specific vegetables (Mills et al. 1989, Giovannucci et al. 1995). A recent study from the Netherlands (Schuurman et al. 1998) found no overall association with vegetable intake. Thus, at the present time, evidence in support of a beneficial role of fruits for prostate cancer is very limited, and the data are inconsistent. While individual nutrients have been studied for their effects on risk, data from the European Prospective Investigation into Cancer and Nutrition (EPIC) showed no association between total fruit and vegetable intake and prostate cancer risk among 130,000 men (Key et al. 2004).

Results from several case-control and cohort studies on fat intake and risk of prostate cancer have been inconsistent. Evidence from many case-control studies has generally found an association between dietary fat and prostate cancer risk (Rose et al. 1986, Kolonel et al. 1988), although some studies have not uniformly reached this conclusion (Schuurman et al. 1999, Giovannucci et al. 1995, Mettlin et al. 1989, Severson et al. 1989). In a recent review of the topic, 11 of 17 case-control studies showed a positive association between prostate cancer and fat intake; none showed a negative association (Yip et al. 1999). In a review of published studies of the relationship between dietary fat and prostate cancer risk, among descriptive studies, approximately half found an increased risk with increased dietary fat and half found no association (Zhou and Blackburn 1997). In general, fat of animal origin seems to be associated with the highest risk (Optenberg et al. 1995, Rose et al. 1986). In a series of patients with prostate cancer, the risk of cancer progression to an advanced stage was greater in men with a high fat intake (Bairati et al. 1998). In another study of men in Hawaii, a significant association was seen between prostate cancer mortality and dietary fat in men over 70 years old (Cole and Rodu 1996, Wynder and Cohen 1997). The announcement in 1996 that cancer mortality rates had fallen in the United States prompted one suggestion that this may be due to decreases in dietary fat over the same time period (Cole and Rodu 1996, Wynder and Cohen 1997). In the Health Professionals Follow-up Study, a positive association was seen between intake of red meat, total fat, and animal fat, and the incidence of prostate cancer (Giovannucci et al. 1993). A recent investigation was from Saudi Arabia (Hanash et al. 2000) and this study showed no relationship between fat intake, mainly from meat and dairy products and prostate cancer risk. The authors of that study did comment that fiber, cereals, cooked tomatoes, rice, tea, fruits and vegetables, among other dietary items that are low in fat, are consumed in large quantities in the average Saudi diet. These healthy items could have been responsible for the lack of an association between fat and prostate cancer.

It is difficult to draw firm conclusions from these epidemiology studies, because of the greater potential for

recall bias, measurement bias and confounding. Therefore, large-scale prospective studies are still required to shed some light on this issue.

Studies in some animal models (Wang et al. 1995, Pollard and Luckert 1986), conducted to examine the relationship between prostate cancer and diet, have found an inhibition of tumor growth with a lower fat intake, or an increased growth with a high fat intake. However, other animal studies that have ensured isonutrient intakes (Pour et al. 1991, Clinton et al. 1997) have not been able to effect the growth of transplanted prostate tumors, or the induction of such cancer by increasing dietary fat. In another extensive animal study (Mukherjee et al. 1999) found that cancer growth was independent of the percentage of fat in the diet, as long as the total energy intake was restricted. The reduction observed in cancer growth was actually similar in all types of energy-restricted laboratory animals. These experiments suggest an overall reduction in energy intake, and not just in fat per se, is the best method to reduce the risk or progression of prostate cancer (Bosland et al. 1999). These studies also suggest that fat in combination with some other unknown dietary factor(s) may be responsible for increasing tumor growth. Even though not significant, the present study indicated with an elevated risk for prostate cancer for those who consumed more than 2 kg oil/fat in a month compared to those who consumed less than 1 kg oil/fat in a month, a finding supporting many of the previous studies on fat consumption and prostate cancer risk.

The explanation for this possible association between prostate cancer and dietary fat is unknown. Several hypotheses have been advanced including:

1. Dietary fat may increase serum androgen levels, thereby increasing prostate cancer risk. This hypothesis is supported by observations from South Africa and the United States that changes in dietary fat change urinary and serum levels of androgens (Hill et al. 1979, Hamalainen et al. 1984).
2. Certain types of fatty acids or their metabolites may initiate or promote prostate carcinoma development. The evidence for this hypothesis is conflicting, but one study suggests that linoleic acid (omega-6 polyunsaturated fatty acid) may stimulate prostate cancer cells while omega-3 fatty acids inhibit cell growth (Rose and Connolly 1991).
3. An observation made in an animal model is that male offspring of pregnant rats fed a high-fat diet will develop prostate cancer at a higher rate than animals fed a low-fat diet (Kondo et al. 1994). This observation may explain some of the variations in prostate cancer incidence and mortality among ethnic groups. An observation has been made that first trimester androgen levels in pregnant blacks are higher than those in whites (Henderson et al. 1998).

Much epidemiologic and case-controlled evidence suggests that diet may be a modifier of prostate cancer risk. Nutrition is apparently a major risk factor for the development and progression of prostate cancer. Based on experimental studies and epidemiologic data mainly from case-control studies or cohort studies, there is strong evidence that reduction of the total energy consumption, a

diet comprising less than 30% fat, and increased intake of phytoestrogens, vitamins D and E and selenium could yield a decreased prostate cancer incidence. The traditional Mediterranean diet has many of the right elements (Meister et al. 2002).

The term 'the Mediterranean diet' was first popularized by Ancel Keys in his book *How to Eat Well and Stay Well: the Mediterranean Way*, in 1975. This followed the publication of his studies which showed that Mediterranean countries have diets associated with low incidence of cancer and cardiovascular disease.

There is now little doubt that the Mediterranean countries enjoy a low risk of many of the diet-related diseases of affluence (Hill and Giacosa. 1992, Hu. 2003).

The Mediterranean is a large area with many different diet patterns, but they are all characterized by high consumption of fruit, vegetables, legumes and dietary fibre and low intakes of meat and saturated fats. In all of these respects they agree with the current concepts of a 'healthy diet' and one towards which many countries in northern Europe are moving (Trichopoulou 2001).

Various factors may contribute to some of the inconsistencies in the association of fruits and vegetable intakes, oil/fat consumption and prostate cancer, including nature of the studies, insufficient sample size, measurement bias, recall bias, and heterogeneity of prostate cancer. The type of fruits and vegetables consumed by cases and controls may vary. The changing life style and dietary habits of people are very complex and vary from person to person. It is difficult to ascertain whether the estimates obtained for one factor is an absolute estimate or confounded by many other factors directly or indirectly. It is difficult to ascertain due to several limitations whether the overall estimates are reliable for the observed effect. The evidence for the estimates obtained from this study may not be a strong one, however, these results may give clues for further investigations and so prospective studies with good design and conduct are required for a better understanding of the role of fruits and vegetables and oil/fat in prostate carcinogenesis.

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