



Your Health Matters

Nutrition & Prostate Cancer

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Scientific evidence suggests that differences in diet and lifestyle may account in large part for the variability of prostate cancer rates in different countries [1].

Good nutrition may reduce the incidence of prostate cancer and help reduce the risk of prostate cancer progression. There are many studies currently being conducted to help further understand how diet and prostate cancer are related. We do know, however, that improved nutrition reduces risk of heart disease, diabetes, and obesity, and usually improves overall quality of life. It is estimated that one-third of cancer deaths in the U.S. can be attributed to diet in adulthood, including diet's effect on obesity [2]. Additionally, a healthy diet helps to increase energy levels, facilitate recovery, and enhance the immune system.

Guidelines for a Healthy Diet

- Plant-based diet
 - Plenty of fruits and vegetables
 - High fiber – whole grains and beans/legumes
- Low fat diet with emphasis on healthy fats
- Limit processed and refined grains/flours /sugars
- Drink plenty of fluids
- Be physically active to help achieve and maintain a healthy weight

Plant Based Diet

A lifelong commitment to a plant based diet may lower a man's risk of developing prostate cancer and may also affect the progression of the disease. Preliminary evidence indicates dietary and lifestyle changes led to a 4% decrease in PSA and significantly decreased prostate cancer cell growth [3]. PSA increased 6% in the control group. A study assessing

* All words with an asterisk (*) are defined in the glossary on pages 33-34

the recurrence of prostate cancer found that a plant based diet, in combination with stress reduction may significantly slow disease progression [4]. PSA doubling time, the hallmark of recurrent prostate cancer, increased from 11.9 months (prestudy) to 112.3 months (intervention). Additionally, individuals who made comprehensive lifestyle changes improved their quality of life [5].

Fruits and Vegetables

- Contain vitamins, minerals, and fiber as well as various cancer-fighting phytonutrients* (examples: carotenoids, lycopene, indoles, flavonols).
- Vibrant, intense COLOR is one indicator of phytonutrient* content.
- There is extensive and consistent evidence that high fruit and vegetable intakes are associated with decreased risks of many cancers [3-18], and while results for prostate cancer risk are not yet conclusive, they are promising [3-6, 18-22].
- Men who consumed at least 28 servings of vegetables per week had a reduced risk of prostate cancer compared with those who ate fewer than 14 servings per week [20].
- The risk for prostate cancer was reduced significantly in men who consumed 2-3 kg (50% lower-risk) and more than 3 kg (60% lower risk) of fruits and vegetables per week compared with men who consumed less than 2 kg per week [5].
- The rate of PSA rise decreased significantly with the adoption of a plant based diet [18]. Men increased their consumption of vegetables from 2.8 servings/day at baseline to 5.0 and 4.8 servings/day at 3 and 6 months, respectively.
- There is some evidence that vegetables, particularly cruciferous vegetables, such as broccoli, cauliflower, cabbage, kale, Brussels sprouts, collard greens, radish, watercress, and bok choy have been associated with a reduced risk of prostate cancer [20-21, 23-26].
 - Men who ate three or more servings of cruciferous vegetables per week had a 41% decreased risk of prostate cancer compared with men who ate less than one serving per week [20].
- Significantly reduced rate of tumor growth was observed when rats were fed a diet rich in tomatoes (34% decrease), broccoli (42% decrease), and a combination of tomatoes and broccoli (52% decrease) [26].
- The anticancer properties of cruciferous vegetables may be due to one of its compounds, diindolylmethane (DIM). DIM may reduce the risk of prostate cancer recurrence [27].
- The benefit of fruits and vegetables in regards to cancer protection may be related to high amounts of carotenoids in certain fruits and vegetables, according to some key population studies [21, 24, 28-35].
- Consume at least five, preferably eight to ten, servings of fruits and vegetables daily for their cancer protective effects [36].

One serving equates to:

- ½ cup fruit or vegetable
- 1 cup raw leafy greens

- ¼ cup dried fruit or vegetable
- 6 fl oz fruit or vegetable juice

Organic Produce

- Organic fruits and vegetables have fewer pesticides, lower levels of total pesticides, and less overall pesticide toxicity than fruits and vegetables grown with chemicals. Although more research is needed, recent evidence indicates a significant increase in antioxidants* in organic and sustainably grown foods versus conventionally grown foods [37-41].
 - Organic vegetables contained a greater concentration of phytonutrients* (phenolic acids) when compared to conventionally grown vegetables [40,41].
- Consuming organic foods appears to increase salicylic acid, which may contribute to a lower risk of cancer [40].
- Choosing organic produce will help you reduce your levels of pesticide exposure and will most likely increase your phytonutrient* consumption.
 - Although washing and peeling your non-organic fruits or vegetables may help to reduce pesticide residues, it will not eliminate them.
- Listed below are produce with the most and least pesticide contamination, both in terms of number of pesticides used and the level of pesticide concentration on an average sampling. Thus, for the fruits and vegetables shown on the most contaminated list, it is wise to buy organic. Alternatively, if organic choices are not available, you may want to consider substituting with produce that tends to contain the least amount of pesticides.

Produce most contaminated by pesticides:	Produce least contaminated by pesticides:
Peaches	Avocados
Strawberries	Corn
Apples	Onions
Nectarines	Asparagus
Spinach	Cauliflower
Celery	Peas (sweet)
Potatoes	Bananas
Bell peppers	Kiwi
Pears	Mango
Cherries	Papaya
Raspberries	Pineapple
Imported grapes	Broccoli

**Adapted from Environmental Working Group – A Shopper’s Guide to Pesticides in Produce

- It is most important, however, to eat fruits and vegetables – organic or conventional. If the availability or cost of organic produce is a barrier, you may wish to avoid those fruits and vegetables that have the highest pesticide residue content.

Fiber – A plant-based diet is naturally high in fiber

- A diet rich in natural fiber obtained from fruits, vegetables, legumes, and whole-grains (for example: whole-grain cereals and breads) may reduce cancer risk and reduce the risk of prostate cancer progression.
- Fiber binds to toxic compounds and carcinogens, which are then later eliminated from the body [42].
- Various mechanisms have been proposed for the protective effects of dietary fiber against cancer. These include:
 - Increased fecal bulk and decreased intestinal transit time, which allow less opportunity for fecal mutagens to interact with the intestinal epithelium [43].
 - Binding to bile acids, which are thought to promote cell proliferation [44].
 - Fermentation in the gut, producing short-chain fatty acids (SCFA). SCFA improve the gut environment and may provide immune protection beyond the gut [43,44].
 - Additionally, whole grains are rich in antioxidants*, including trace minerals and phenolic compounds, which have been linked to disease prevention [44].
- A high fiber diet works to reduce hormone levels that may promote prostate cancer progression [43, 45-46].
- Some research indicates an inverse relationship between prostate cancer and dietary fiber intake [46, 47] or fiber-rich foods, such as whole grains, legumes, nuts, and seeds [21, 24, 48].
- One study indicated that a high fiber, low-fat diet followed for only 10 days resulted in serum changes that reduced the growth of prostate cancer [46].
- Fiber may [45] or may not [49] lead to a reduction in PSA values. A recent study assessing the rate of PSA rise found that the rate decreased significantly when men increased their consumption of whole grains [18]. Median intake of whole grains increased from 1.7 servings/day at baseline to 6.9 and 5.0 servings/day at 3 and 6 months, respectively.
- Prostate cancer mortality is inversely associated with consumption of cereals and nuts/seeds, according to a study in the Journal of the National Cancer Institute [50].
- A high-fiber diet is also associated with less obesity [51].
- Aim for 30-35 grams of fiber daily [52].
 - Choose breads with three or more grams of fiber per slice.
 - First ingredient on the label should be whole or sprouted grain flour, not white flour or unbleached white flour, or enriched wheat flour.
 - Whole grains include oats, barley, brown rice, quinoa, amaranth, bulgur, millet, spelt, soba noodles, etc.
- Refer to the high-fiber sources table for more information (see page 24).

SUGARS AND THE ROLE OF INSULIN

- High sugar foods are usually highly processed and refined, low in nutrient value, and low in dietary fiber.
- Furthermore, these foods appear to increase serum insulin* and serum insulin-like growth factor (IGF-I) levels, which lead to both the development and promotion of cancer [53-61].
- Sugars to be consumed in limited amounts include products made with refined flours (for example: white bread, white rice, white pasta) or refined grains, alcohol, and sweets, such as candy, cookies, cakes, and pies.

Sugars and Insulin - Bottom Line

- To help control your insulin level:
 - Eat a high fiber diet with limited refined and processed foods
 - Follow a low fat diet
 - Exercise
 - Maintain a healthy body weight

LOW FAT DIET

- The increased cancer risk observed in developed countries may be, in part, due to the fact that a high fat diet stimulates increased testosterone levels, which is known to be associated with prostate cancer growth [62-63].
- A comprehensive review reported that 20 of 30 studies found positive, although not all statistically significant, associations between dietary fat intake and prostate cancer risk [64].
- While a positive association between prostate cancer and fat intake was not observed in all studies [65], some prospective studies did report significant findings [66-69].
- Prostate cancer mortality has also been associated with dietary fat [70]. Strong correlations were noted for meat, added fats and oils, ice cream, margarine, salad/cooking oil, and vegetable shortening.
- Most researchers agree to aim for 20% of your total calories from fat, with less than 8% of total calories from saturated fat [52].
- The type of fat is significant and may, in fact, be of greater importance than total fat.

Saturated Fats

- Several studies indicate a positive association between saturated fat intake from meat and dairy products (animal sources) and prostate cancer [28, 71-74]. Eating red meat [66, 69, 75-76] and dairy products [28, 66, 75-76] also appear to be related to an increased risk of metastatic prostate cancer.

- Reduce or eliminate consumption of meats and whole milk dairy products, including cheese.
 - Cheese is typically between 60-80% fat, much of which is saturated fat.
 - Be advised, however, that low fat and non-fat dairy have also been associated with prostate cancer (see p. 19-20).
- Limit use of butter, mayonnaise, baked goods, and regular salad dressings due to high saturated fat and total fat content.
 - Consider rice vinegar, balsamic vinegar, lemon juice, or salsa as alternative salad dressings.

Trans Fatty Acids

- Trans fatty acids, or hydrogenated oils, are known to be atherogenic, increasing one's risk of heart disease [77-80].
- Preliminary research indicates these fats may also be associated with increased cancer risk [17, 81-82].
- A recent prostate cancer trial reported a 30% increased cancer risk in men who used margarine once or more daily [19].
- Avoid hydrogenated fats, such as margarine, fried foods, commercial peanut butter, salad dressings and processed foods including breads, crackers, cereals, and cookies that are high in harmful trans fatty acids.
 - When you read that a product contains "hydrogenated" or "partially-hydrogenated" oils, consider putting it back on the shelf. (Be sure to bring your reading glasses when shopping.)
 - Trans fatty acid labeling came into effect in January 2006, the amount of these fats in a product must now be clearly identified.

Omega-9 Fatty Acids

- Most research at this time indicates a neutral relationship [19, 65, 83-84], or a slightly protective effect [85-86] between these fats (and foods that are rich in these fats) and risk of prostate cancer.
- Dietary sources include extra-virgin olive oil, canola oil, macadamia nut oil, almonds, and avocados.
- Nuts are generally concentrated in omega-9 fatty acids, but remember they are high in fat.
 - Minimize consumption of nuts to no more than ¼ cup with meal or snack to limit total fat and calories.
 - More research is necessary, but some studies have shown nuts to have a protective effect against prostate cancer [85].

Essential Fatty Acids (EFA)

Essential fatty acids are necessary for the formation of healthy cell membranes, the proper development and functioning of the brain and nervous system, and for the production of hormone-like substances called eicosanoids* (thromboxanes, leukotrienes, prostaglandins). Among other body functions, these chemicals regulate immune and inflammatory responses.

Eicosanoids* formed from the omega-6 fatty acids have the potential to increase blood pressure, inflammation, platelet aggregation, allergic reactions and cell proliferation. Those formed from the omega-3 fatty acids have opposing affects. Current research suggests that the levels of essential fatty acids and the balance between them may play a critical role in the prevention and treatment of cancer.

Omega-6 Fatty Acids

- Although not all studies [69, 87-89] have observed an association, a high intake of omega-6 fatty acids (linoleic acid, which can be converted to arachidonic acid) may stimulate the growth of prostate cancer cells [83, 90-94].
- Preliminary evidence suggests that the balance between omega-6 fatty acids and omega-3 fatty acids can affect prostate tumors [92-93]. Thus, the ratio between omega-6 and omega-3 fatty acids may be of paramount importance. It appears optimal to achieve a 1:1 to 4:1 ratio.
 - Dietary sources of omega-6 fatty acids include meat, butter, egg yolks, whole milk, corn oil, safflower oil, sunflower oil, and cottonseed oil.
 - Substitute olive, almond, macadamia nut, or canola oil for your current cooking oil or fat. These oils are rich in omega-9 fatty acids, which do not appear to increase cancer risk.

Omega-3 Fatty Acids

- Omega-3 fatty acids [(alpha-linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA)] may reduce risk for prostate cancer as well as reduce the risk of cancer progression [19, 84, 93-97].
- Additionally, these fats help strengthen the immune system.
- Mechanisms proposed for their protective effects include:
 - Induce apoptosis* (cell death)
 - Suppress cancer cell initiation
 - Compete with arachidonic acid, which limits harm from arachidonic acid
- In vitro and animal studies have consistently reported reduced cell proliferation and decreased rate of cancer progression with omega-3 fatty acids [94, 98-100].
- Men who consumed fish 3 or more times per week also had a 44% lower risk of prostate cancer, especially for metastatic prostate cancer where the effect was even greater [96].

- Consuming fish 2 or more times a week was also associated with a reduction in prostate cancer progression [101].
- Researchers in New Zealand reported that men with high levels of EPA and DHA, the omega-3 fats found in fish, had a 40% lower risk of prostate cancer than those with low blood levels [95].
- A 30-year follow-up study found that men who ate no fish had a 2 to 3 times higher frequency of prostate cancer than those who ate moderate or high amounts of fish [97].
- Dietary sources include cold-water fish (for example: salmon, trout, herring, sardines, mackerel, sablefish), flaxseeds, walnuts, pumpkin seeds, soybeans, and canola oil.
 - It may be wise to consume fish at least twice weekly to obtain an adequate amount of omega-3 fatty acids.
 - Alternatively, incorporating one of the following foods on a regular basis will help to achieve sufficient omega-3 fatty acids:
 - 1-2 Tbsp ground flaxseed
 - 1 oz walnuts
 - ½ - 1 cup cooked soybeans
- Fish and plant-based foods, however, contain different types of omega-3 fatty acids.
 - Fish contains EPA and DHA, two specific fatty acids that have shown promising results in protecting against cancer [102].
 - The plant-based omega-3 fatty acid sources, such as flaxseed and others listed above, contain ALA. In an ideal environment, ALA is converted to EPA and DHA, however, this process is inefficient [103-104]. On the positive side, the conversion process is enhanced by following a diet that is low in saturated fats and low in omega-6 fatty acids [105].
 - Although not all studies agree [87, 106], some research indicates a positive association between ALA and prostate cancer [69, 76, 89-90, 107]. It should be noted, however, that the primary source of ALA in these studies was red meat, milk, butter, mayonnaise, and margarine [69, 76, 107].
- If you're considering an omega-3 fatty acids supplement, choose one that is highest in EPA and DHA concentration.

Fat – Bottom Line

- Less fat is better.
- Limit animal fat.
- Avoid hydrogenated fats.
- Extra-virgin olive oil or canola is preferred for salads and cooking.
- Increase omega-3 fatty acids.

ADEQUATE FLUIDS

- The functions of water in the body include the following:
 - Carries nutrients and waste products.
 - Participates in chemical reactions.
 - Acts as a lubricant and cushion around joints.
 - Acts as a shock absorber in the eyes and spinal cord.
 - Aids in the body's temperature regulation.
 - Maintains blood volume.
- Increased fluid intake is needed for a high fiber diet.
- Drink plenty of water daily to help meet fluid needs.

CALORIC INTAKE

- Substantial evidence indicates that a high caloric intake increases one's risk of various cancers, including prostate cancer [22, 73, 106, 108-110].
- A case-control* study reported a 115% increased risk in local prostate cancer and a 96% increased risk in regional/distant prostate cancer for those consuming higher calorie diets (2439 or more calories/day) compared with individuals consuming a lower calorie diet (less than 1322 calories/day) [109].
- In a separate study, researchers reported a nearly four-fold increase in prostate cancer risk in men who consumed the most calories (more than 2624 calories/day) compared with men who consumed the least calories (1064 calories/day) [110].
- An earlier study found that caloric intake was positively associated with preclinical prostate cancer risk; as caloric intake increased, cancer risk rose significantly [73]. The greatest risk was for men who consumed more than 3475 calories/day.
- The mechanism involved may be related to the decrease in IGF-I observed when caloric intake is restricted [73, 111].

BODY MASS

- Higher body mass and physical inactivity may contribute to prostate cancer risk.
 - A large prospective study observed a significant positive association between body mass index (BMI) and prostate cancer risk [65].
 - A cohort* study reported obese men to have a 20% increased risk of dying from prostate cancer and those men who were severely obese had a 34% elevated risk [112].
 - This research was further supported by recent evidence that obesity is a risk factor for aggressive prostate cancer [113-115].

- The risk of prostate cancer may be reduced with weight loss [115]
- According to a UCSF study with over 2,000 men, men who maintained a healthy body weight were less likely to have a recurrence [116].
 - Obese men had a 30% increased risk of cancer recurrence, compared with those with lower body weights. Very obese patients (BMI >35)¹ heightened their risk of recurrence by 69%.

PHYSICAL ACTIVITY

- A prospective study suggested that men in the highest category of exercise reduced their risk of advanced prostate cancer by 36% compared to the non-exercisers [117]. The risk of dying from prostate cancer was reduced by 33%.
- Researchers conducted an 11-day study where men followed the Pritikin program [46]. The Pritikin program focuses on a diet that is high in vegetables, fruits, whole grains, and very low in fat in combination with 60 minutes of daily exercise.
 - Serum IGF-I decreased by 20% after following the program for 11 days. This reduction in IGF-I may be related to the lower serum insulin* levels observed via a healthy low fat plant-based diet and regular exercise.
- These findings were further substantiated by researchers who placed blood serums of three groups of men (Pritikin followers of 14 years, regular exercisers of 14 years with no modified diet, and sedentary overweight men who consumed a high-fat, high-sugar diet) in culture dishes containing prostate cancer cells [54].
 - In a mere three days, researchers reported significant change in the cells. The blood serum of the Pritikin followers destroyed 50% of the prostate cancer cells compared to the exercise-only group where approximately 25% of the cells were destroyed and less than 3% of the cancer cells were destroyed in the overweight, sedentary men eating a more typical high-fat American diet.
- Healthy weight control is encouraged with an emphasis on exercise to preserve or increase lean muscle mass.

Foods, Nutrients, & Food Components of Special Interest to Cancer Survivors

ANTIOXIDANTS – Found in abundance in fruits and vegetables, although vitamin E may need supplementation.*

- Prevent oxidative damage in body cells.
 - Research indicates a link between oxidant damage and prostate carcinogenesis.
- Examples of antioxidant* nutrients and nonnutrients include vitamins A, C, and E, selenium, lycopene, and beta-carotene.

¹ BMI is calculated by weight wt(kg)/ht²(m²)

- Note that patients may be advised to NOT consume high dose antioxidant* supplements during chemotherapy or radiation treatments. Antioxidant* consumption via food sources and a basic multivitamin are very safe.

Lycopene

- Antioxidant* that scavenges free radicals to reduce tissue damage. Evidence suggests that one daily serving of tomatoes or tomato products, the richest source of lycopene, helps protect against DNA damage [118].
- Higher blood levels of lycopene are associated with reduced risk of prostate cancer [33, 119-121].
- Furthermore, lycopene inhibits the multiplication of prostate cancer cells [122-123].
- Men who consumed tomato sauce 2-4 times weekly had a 34% lower risk for prostate cancer in the largest prospective study to date [32].
- Thirty-two men with prostate cancer consumed 30 mg daily lycopene in the form of tomato sauce for 3 weeks; researchers reported a 28% reduction in oxidative DNA damage and a 17.5% decrease in PSA values [124-125].
- In a smaller study where men consumed 30 mg lycopene daily for three weeks, men had 18% lower PSA levels whereas the control group had a 14% increase in PSA levels [122].
- The consumption of tomato sauce 2 more times per week decreased the risk of prostate cancer progression [101].
- Significantly reduced rate of tumor growth was observed when rats were fed a diet rich in tomatoes (34% decrease), broccoli (42% decrease), and a combination of tomatoes and broccoli (52% decrease) [26].
- Dietary sources include tomato products, such as tomato juice, V-8 juice, spaghetti sauce, and ketchup as well as guava, grapefruit, papaya, and watermelon.
 - Cooked tomato products or juices contain higher amounts of lycopene.
- Additionally, lycopene-rich foods are best absorbed in the presence of fat (such as a small amount of olive oil) [126].
- As with most nutrients and non-nutrients, it is best to obtain lycopene from foods. Supplements appear to be capable of interfering with normal nutrition by promoting oxidative damage, which increases cancer risk [127].
- In western countries, tomato-based products comprise 85% of dietary lycopene [128].
- How much do I need? Research indicates positive changes with 30 mg lycopene daily, which would be approximately:
 - 4 ½ tbsp tomato paste
 - ¾ cup tomato sauce
 - 12 fl oz tomato juice
 - 8 medium raw tomatoes
 - 4 cups watermelon

Selenium

- Antioxidant*, which scavenges free radicals and suppresses damage due to oxidation in the tissues. Also is essential for the immune system.
- Selenium appears to inhibit cellular changes that may lead to prostate cancer. Selenium may also work to inhibit angiogenesis* [129] and induce apoptosis* or cell death [130].
 - Angiogenesis may be defined as the growth of new blood vessels. In cancer, new vessels allow tumor cells to escape into the circulation and lodge in other organs (tumor metastases) as well as enable a tumor to grow.
- Selenium has consistently been reported to reduce risk of prostate cancer [131-134].
- Low plasma selenium is associated with a 4 to 5-fold increased risk of prostate cancer [135].
 - Note that plasma selenium decreases with age, thus obtaining sufficient selenium may be particularly beneficial to older men.
- Additionally, selenium supplements have been shown to decrease the recurrence of prostate cancer by 63% [131].
- Some research indicates that the combination of selenium and vitamin E may work synergistically in reducing risk for prostate cancer [136]. This combination is currently being reviewed in the Selenium and Vitamin E Chemoprevention Trial (SELECT) [137].
- Selenium is a precursor to the glutathione* (GSH) antioxidant* system. GSH is the principal protective mechanism of the cell and is a crucial factor in the development of the immune response by the immune cells [138].
- Dietary sources include Brazil nuts, seafood, enriched brewer's yeast, and grains. Selenium content depends somewhat on the amount in the soil in which the products are grown.
 - A mere two Brazil nuts will provide more than 200 mcg selenium.
- Most research studies have used a 200 mcg supplement daily [130, 139].
 - Selenomethionine is a type of selenium supplement that appears to have greater bioavailability than other forms [140-141].
 - Selenium supplements should be taken with food.

Nutrient	Recommended Dietary Allowance (RDA)	Prostate Cancer Research	Tolerable Upper Intake Level (UL)
Selenium	55 mcg	200 mcg	400 mcg

Vitamin A and Beta-Carotene

- Beta-carotene is one of the 600 carotenoids that can be partially converted into vitamin A in the body.
- Beta-carotene is an antioxidant* that may play a role in helping prevent various diseases, including some cancers.

- Some research indicates that dietary beta-carotene may reduce prostate cancer if combined with a diet rich in fruits and vegetables, and low in fat [24, 33-35].
 - A study in Japan found beta-carotene in vegetables to be significantly protective, but the effect was not observed in fruits [35].
 - Plasma carotenoids were associated with lowering risk of prostate cancer, but no association was observed between carotenoids and disease progression [142].
 - Other studies, including a case-control* study in Canada and a 30-year follow-up study, observed no association between consumption of beta-carotene and prostate cancer [33, 143].
 - Beta-carotene supplements have not been shown to have any beneficial properties in regards to prostate cancer [144-146].
 - Furthermore, findings from the ATBC Trial revealed a 23% increased risk of prostate cancer incidence in men who consumed 20 mg beta-carotene supplements [147].
- Dietary sources include carrots, winter squash, sweet potatoes, cantaloupe, and mangoes.

Vitamin C

- May prevent the formation of free radicals that cause DNA damage and help repair damaged DNA.
- No consistent relationship between vitamin C and prostate cancer. Some studies report slight reductions in risk [22, 76, 148, 149] while others have found no effect [143].
 - Case-control* studies in Spain [76] and Uruguay [22] that used food frequency questionnaires reported an inverse association of vitamin C and prostate cancer.
 - An additional case-control* study reported a 23% reduction in prostate cancer risk from daily vitamin C use [148].
 - An in-vitro study observed a protective effect of vitamin C against prostate cancer [149].
 - Inhibits proliferation and survival of tumor cells.
 - A large cohort* study, however, found no protective effect for vitamin C against prostate cancer [144].
 - Additionally, a 30-year follow up study observed no association between consumption of vitamin C and prostate cancer [143].
- Dietary sources include various fruits and vegetables:
 - Fruits: papaya, citrus fruits, kiwi, cantaloupe, mango, strawberries
 - Vegetables: yellow and red peppers, broccoli, tomatoes
- Supplements containing 250-500 mg taken once or twice daily appear safe.
- Some evidence suggests higher dosages (more than 1000 mg daily) may actually promote oxidative damage. There are, however, other studies that have not observed adverse effects with vitamin C supplementation.

Nutrient	Recommended Dietary Allowance (RDA)	Prostate Cancer Research	Tolerable Upper Intake Level (UL)
Vitamin C	90 mg (males)	NA	2000 mg

Vitamin E

- Vitamin E acts as a cellular antioxidant* and an anti-proliferating agent. It consists of both tocopherols and tocotrienols.
- Studies show vitamin E may reduce risk of prostate cancer and inhibit prostate cancer cell growth [22, 147, 150-152].
 - Cancer incidence reduced by 33% and death from prostate cancer reduced 41% in a 6-year follow-up study where men consumed 50-100 IU vitamin E daily [147].
- Vitamin E may reduce the incidence of prostate cancer, and also lower the rate of conversion from latent to aggressive forms of prostate cancer.
- Results suggest that long-term vitamin E (alpha-tocopherol) supplementation decreases serum androgen concentrations, which is related to a reduced incidence of and mortality from prostate cancer [134].
- Researchers reported lower serum gamma-tocopherol (a form of vitamin E) values in men with prostate cancer in a recent cohort study [151]. Furthermore, large doses of alpha-tocopherol suppress levels of gamma-tocopherol [153-155].
- The most provocative study found that men with the highest plasma gamma-tocopherol concentrations had a significantly fivefold lower risk of prostate cancer compared with men in the lowest quintile [150]. This effect was not significant for plasma alpha-tocopherol concentrations.
- Other researchers have also found gamma-tocopherol to offer a protective effect against prostate cancer [151-153, 156].
- Dietary sources of vitamin E include vegetable oils, wheat germ, nuts, seeds, soybeans, sweet potatoes, and avocado. Due to the high fat content of many dietary sources, a supplement may be beneficial.
- Many studies used a 400 IU vitamin E supplement daily, however, a multi-vitamin may easily provide 50-100 IU vitamin E.
- There has been considerable discussion regarding the different forms of vitamin E.
 - Natural forms of vitamin E (gamma-tocopherol, d-alpha-tocopherol) appear to be better absorbed by the body, but are more expensive [157-158]. A supplement containing mixed tocopherols (d-alpha, gamma, beta) and tocotrienols is preferred. The combination of gamma- and alpha-tocopherol may offer greater protection from DNA damage than alpha-tocopherol alone [159].
 - Many of the studies on vitamin E and prostate cancer have used synthetic forms of vitamin E (dl-alpha-tocopherol) [134, 147, 157, 160]. A recent study, however, reported that the natural form of vitamin E was more effective in the suppression of prostate cancer, and at a lower concentration [157]. More specifically, vitamin E succinate may offer these protective effects [161].

- Although more research is needed to determine the recommended dosage, it may be suggested to take 200IU alpha-tocopherol with 400mg gamma-tocopherol.

Nutrient	Recommended Dietary Allowance (RDA)	Prostate Cancer Research	Tolerable Upper Intake Level (UL)
Vitamin E	15 mg 33 IU dl-alpha (synthetic) 22 IU d-alpha (natural)	50-200 IU *	1000 mg 2222 IU dl-alpha 1493 IU d-alpha

* Vitamin E form varies among studies

FLAXSEED

- Flax is a good source of omega-3 fatty acids and fiber, contains protein, calcium, potassium, B vitamins, iron, and boron.
- Flax may also work to block tumor growth, inhibit angiogenesis*, and enhance immune function [162].
- Lignans*, phytoestrogens found in flax, appear to bind with testosterone, which lower circulating levels of testosterone [163-164]. This action may be one of the protective mechanisms of flax.
- Preliminary data suggest reduced tumor growth and spread with the use of flaxseed [163, 165-166].
- One study reported that prostate cancer patients following a 20% fat diet and consuming 30 gm flax daily (2 ½ Tbsp) resulted in reduced cancer growth rates the more days patients followed the above diet [157].
- Animal data indicates that flaxseed may lead to less aggressive tumors and a lower risk of metastasis [165].
 - Flax can have a laxative-like effect, thus, it is wise to gradually increase consumption.
- Ground flax seeds have greater bioavailability than whole flax seeds. Flax seeds may easily be ground in a coffee grinder, blender, or food processor.
- Ground flax seeds can be sprinkled into many foods and beverages, including hot cereals, tomato sauce, fruit smoothies, brown rice or other grains, and more.
- Due to the instability of these fatty acids, it is best to store flax in the refrigerator or freezer.
- Note: Flax seed OIL is highly concentrated and lacks the protein, fiber, vitamins, minerals, and lignans that are found in ground flax seeds. The use of ground flax seed is highly preferred.
- Dosage: 2 tablespoons ground flaxseed daily.

GARLIC (Allium Family)

- Many studies indicate that in populations that consume high amounts of garlic, scallions, onions, leeks, and shallots (all members of the same plant family) there is a reduced rate of overall cancer [19, 34, 167-168].
 - These allium vegetables are rich in flavonoids and organosulfur compounds that have anti-cancer properties.
- A Japanese study found that men who consumed 1/3 oz. or more of one of the allium-rich foods mentioned above had approximately 50% reduction in prostate cancer risk compared with men who consumed lesser amounts [167].
- One study reported a reduced risk of prostate cancer when subjects consumed natural garlic at least twice weekly [34].
- According to the prostate cancer literature, garlic supplements do not appear to have the same beneficial effects as real garlic, either raw or cooked [169].

GENOTOXINS: HETEROCYCLIC AMINES (HCA's) & POLYCYCLIC AROMATIC HYDROCARBONS (PAH's)

- Natural components in meat, such as amino acids, creatine*, and polysaccharide precursors, are converted to HCA's during high-temperature cooking. HCA's are known to cause cancer in laboratory animals [170-173]. While human research is forthcoming [170, 174-177], only one study has observed no association between HCA's and prostate cancer [178].
- It may be that cancer risk is heightened in people who metabolize these compounds more rapidly. Some individuals appear to have genes that encode enzyme production, which readily metabolize HCA's [174, 179].
- Carcinogenic activity of HCA's is affected by various dietary factors [179]:
 - Factors that enhance carcinogenesis* when combined with HCA's include:
 - ❑ High-fat diet
 - ❑ Caffeine
 - Factors that inhibit carcinogenesis* when combined with HCA's include:
 - ❑ DHA
 - ❑ Conjugated linoleic acid (CLA)
 - ❑ Isoflavones
 - ❑ Green tea catechins*
 - ❑ Indole-3 carbinol
 - ❑ Probiotics
 - ❑ Gamma-tocopherol
- Additionally, the type of protein cooked can also affect the concentration of HCA's. It has been reported, for example, that chicken has more than 100 times the number of HCA's than salmon [179]. London broiled steak had more than 600 times the amount of HCA's when compared to salmon.

- The most important variables contributing to the formation of HCA's are [170, 181]:
 - Cooking temperature (greater than 300°F)
 - Cooking time (greater than 2 minutes)
 - Cooking method (frying, oven grilling/broiling, barbecuing)
- Charring of food (charcoal-broiled or smoked foods) contribute to PAH's [182].
- In the western diet, fried meat is the main source of exposure to HCA's.
 - Some research suggests that chicken is the largest source of HCA's.
 - May be related to its frequent consumption.
 - Preparing chicken using a lower-temperature cooking method produces much lower levels of HCA's.
- Meat can potentially be made "safer" to eat by being cooked in a way that does not lead to the formation of these compounds.
 - Choose lean, well-trimmed meats to grill. Lean cuts cause less fat to drip onto the coals (and hence, fewer flame-ups).
 - Using marinades significantly reduces the amount of HCA's.
 - Brief microwave preheating of raw meat, for example, substantially reduces the HCA content.
 - Small portions require less time on the grill.
- Grill vegetables or meat substitutes that do not lead to the formation of HCA's or PAH's.

GREEN TEA

- Tea contains phytonutrients* known as polyphenols (flavonoids) that provide antioxidant* and anti-cancer properties [183].
 - May block the formation of cancer-causing compounds, such as nitrosamines*.
 - Prevents DNA damage [185].
 - May inhibit tumor growth and induce apoptosis* [186-187].
 - Inhibits COX-2 activity [188].
 - Inhibits IGF-I [189]
 - Increase immune response [187]
 - Many studies indicate a lower risk of cancer with green tea consumption, but more research is needed for conclusive evidence [190-193].
- Green tea catechins*, a category of polyphenols*, suppress cell growth and induce cell death in human prostate cancer cells [191, 194-197].
- Animal studies have observed inhibition of prostate cancer development, lower serum testosterone concentrations, and increased survival with green tea [198].
- The combination of soy and green tea synergistically inhibited tumor weight and metastasis and

significantly reduced serum concentrations of both testosterone and DHT [199].

- Nonetheless, the Ohsaki Cohort* study reported no association between green tea and the risk of prostate cancer [200].
- Furthermore, researchers found green tea (6 gm daily) to have no significant effect on PSA levels [165, 201]. The median length of the study, however, was only one month.
- A case-control study observed heightened protective effects of green tea with increasing frequency, duration, and quantity of green tea consumption [190].
- Green tea does naturally contain caffeine although a much lower amount than coffee or black tea. Although decaffeinated green tea is also available, reports suggest that the phytochemical content may be two-thirds less than regular green tea. If you choose a decaffeinated green tea, be sure to choose one that uses a natural decaffeination method, such as water and effervescence.
- A cup of green tea daily would likely be a healthy addition to your diet, but evidence suggests that 3 or more cups are needed for the cancer protective effects [190].

POMEGRANATE (Punica granatum; Punicaceae)

- Research suggests that pomegranate exhibit strong anti-inflammatory and antioxidant* effects [202].
 - Pomegranate seed oil and fermented juice block the cancer cells' oxygen supply, slow cell growth, and promote cell death [203].
- Animal data suggest that pomegranate fruit extract significantly inhibits tumor growth while also significantly decreasing PSA levels [202].
- Prostate cancer patients significantly increased PSA doubling time (mean of 54 months compared to 15 months) by consuming 8 oz pomegranate juice daily [204].

SOY

- Soy contains various nutrients, including protein, fiber, calcium, and B vitamins.
- Soy is rich in antioxidants* known as isoflavones, namely genistein and daidzein.
- Associated with reduced rates of heart disease [205-207], protection against osteoporosis [208-209], and certain types of cancer, including prostate cancer [24, 50, 207, 210-215].
- Soy has been one dietary component thought to play a role in the lower rate of prostate cancer in Asian countries. A recent meta-analysis* found that the consumption of soy foods was associated with a lower risk of prostate cancer [216].
- Research shows that plant estrogens, such as soy, may also prevent prostate cancer by [211, 217]:
 - Decreasing blood androgen levels
 - Increasing SHBG* (sex hormone-binding globulin) concentration
 - Binding to hormone receptors
 - Inhibiting 5-alpha reductase
 - Restricting other enzymes associated with cell growth

- Causing direct tumor destruction that essentially starves the tumor
- Decreasing IGF-I
- Recent studies found that soy flour (120 mg isoflavones daily) [218] and tofu [63] decreased serum testosterone levels in healthy men, further substantiating that soy may protect against prostate cancer.
- A low-fat diet combined with soy has been shown to decrease PSA values significantly in a 3-month period [219]. Soy foods and a low fat diet may have a symbiotic relationship [220].
- A different study, however, found no significant decrease in PSA values in men who consumed 44 gm soy protein for one month [205]. A review study of prostate cancer patients noted that PSA values decreased significantly with soy isoflavones in four of eight trials [221]. Isoflavones in healthy subjects did favorably affect PSA.
- Soy products were found to be significantly protective in a study that included data from 42 countries [50].
- Vegan proteins, such as soy, can be expected to decrease circulating IGF-I activity, which may impede cancer induction [206].
- Dietary sources include soybeans, tofu, tempeh, edamame, miso, soy cheese, soy nuts, soy milk, textured vegetable protein (TVP), and more. Soy supplements or extracts are not recommended.
- Consume one or more servings of soy daily.

Source	Amount of Soy Protein (gm)	Amount of Soy Isoflavones (mg)
Miso (1 Tbsp)	2	7-10*
Soybeans, edamame (1/2 cup)	11	35*
Soy milk (8 fl oz)	10	30*
Soy nuts (1/4 cup)	19	40-50*
Tempeh (1/2 cup)	19.5	36*
Tofu (4 oz)	13	38.8*

* Isoflavone content varies by brand

VITAMIN D and CALCIUM

- Vitamin D is known to inhibit prostate cancer in animals [222-226] and although human research remains unclear, vitamin D appears to be of benefit [132, 227-229]. Vitamin D is believed to be important in the protection of human prostate cells [229-232].
- Epidemiological* studies indicate that sunlight exposure is inversely proportional to prostate cancer mortality and that prostate cancer risk is greater in men with lower levels of vitamin D [227-228, 230, 233-234].
- Increasing serum 25 (OH) -vitamin D levels by 25 nmol/l was associated with a 17% reduction in total cancer incidents and 29% decrease in total cancer mortality [235].

- Optimal serum 25 (OH)-vitamin D levels have not been defined though research indicates 90-100 nmol/l (36-40 ng/mL) may be ideal [250].
- A recent study on advanced prostate cancer reported that the combination of vitamin D and the chemotherapy agent taxotere was twice as effective as taxotere alone based on PSA responses [236].
- Epidemiological* studies (12 of 14) have reported increased prostate cancer incidence with high calcium intakes [132].
 - One theory is that high amounts of calcium (more than 2000 mg daily) suppress circulating vitamin D blood levels [132, 237-238].
- A recent meta-analysis* supported association between high intake of calcium and dairy overall increasing the risk of prostate cancer [239]. Men who consumed 1329-2250 mg of calcium/day had a 39% greater risk of prostate cancer compared to men who consumed 228-802 mg/day.
- The relationship between dairy foods and increased prostate cancer risk may be due to the high calcium content in dairy and/or to the animal fats in dairy [65, 237, 240].
 - Some studies using skim or low-fat milk have found an increased association with prostate cancer, supporting the calcium connection [65, 237, 241].
 - Men who consumed 21 servings of dairy per week, compared to men who consumed 5 or less per week, more than doubled their risk of prostate cancer [241].
 - Dietary calcium (920 mg/day compared to 455 mg/day) was strongly associated with prostate cancer risk.
 - Men who drank more than 6 glasses of milk a week had lower levels of vitamin D than men who drank fewer than 2 glasses of milk a week.
- One analysis identified a four- to fivefold risk elevation with very high (more than 2000 mg daily) or very low calcium intakes (less than 500 mg daily) [132].
- Higher calcium intakes resulted in a modest 7% increase risk of localized prostate cancer, but more than 200% increased risk for advanced prostate cancer compared to men with the lowest calcium intake [109]. These results were further substantiated by a more recent cohort* study [242]. Researchers suggested that 1500 mg/day or more of calcium may increase risk of advanced and fatal prostate cancer.
- Additionally, a positive association between IGF-I and intake of milk, dairy products, and calcium has been reported [243].
- Thus, it is recommended to consume adequate amounts of vitamin D and calcium, but to avoid high calcium intakes (more than 1500 mg daily).
- Non-dairy sources of vitamin D include cold-water fish (for example: salmon, trout, herring, sardines, mackerel), fortified products (for example: soy milk, cereals), and sunlight.
- Ten to fifteen minutes of sunlight 3-4 times per week should provide you with adequate vitamin D [244]. Individuals with darker pigmented skin may need 20-25 minutes of sunlight.
- Vitamin D absorption declines with age, and vitamin D deficiency is not uncommon among older adults [245-249].
- Patients, especially those on hormone therapy, may benefit from a serum vitamin D blood test (25-OH) and a dual energy x-ray absorptiometry (DEXA) bone density scan.

- Vitamin D3 or cholecalciferol is the preferred supplemental form of vitamin D. The bioavailability of this form is significantly greater than vitamin D2 or ergocalciferol.
- Optimal serum 25-OH vitamin D levels have not been defined though research indicates 90-100 nmol/L (36-40 ng/mL) may be ideal [250].
- If supplementing with vitamin D, it is wise to test serum vitamin D. Although unlikely, excessive doses of vitamin D can be toxic and cause high blood levels of calcium.
 - Vitamin D3 or cholecalciferol is the preferred supplemental form of vitamin D. The bioavailability of this form is significantly greater than vitamin D2 or ergocalciferol.

Nutrient	Adequate Intake (AI)	Prostate Cancer Research	Tolerable Upper Intake Level (UL)
Vitamin D	<u>Under 50 yrs:</u> 5 mcg or 200 IU <u>50-70 yrs:</u> 10 mcg or 400 IU <u>Over 70 yrs:</u> 15 mcg or 600 IU	AI	50 mcg or 2000 IU
Calcium	<u>19-50 yrs:</u> 1000 mg <u>Over 50 yrs:</u> 1200 mg	AI	2500 mg

Food Safety

- Especially important for those with weakened or impaired immune systems.
- The following recommendations have been adapted from guidelines provided by the American Cancer Society.
 - Wash foods thoroughly before eating.
 - Keep all aspects of food preparation meticulously clean.
 - Use special care in handling raw meats, poultry, and eggs.
 - Thoroughly clean all utensils, counter tops, cutting boards, and sponges that touched raw meat.
 - Thaw meats and fish in the refrigerator.
 - Do not eat perishable foods that have been left out of the refrigerator for more than two hours.
 - Store foods at low temperatures (less than 40°F) to minimize bacterial growth.
 - When eating in restaurants, avoid foods that may have bacterial contamination, including sushi, salad bars, buffets, unpasteurized beverages or food products, and raw or undercooked meat, poultry, fish, and eggs.

Herbs

MILK THISTLE

- There is no conclusive evidence regarding milk thistle and prostate cancer.
- There have been a few studies (in vitro and in vivo studies only) that suggest the components in milk thistle, silymarin and silibinin, inhibit growth and apoptosis of prostate cancer cells [251-255].
 - The mechanism of action is still to be determined, but may be related to the inhibition of the IGF-I pathway [252, 255].

PC-SPES

- Combination of seven Chinese herbs and one North American herb, saw palmetto.
- This herbal combination exhibits estrogenic activity and can reduce blood testosterone levels, hence its association with reduced prostate cancer risk [256-257].
- FDA recalled PC-SPES supplements in February 2002 because it was found to contain undeclared prescription drug ingredients [256-257].
- This product could cause serious health effects. If you are using PC-SPES, please discuss with your physician.

SAW PALMETTO

- A partially dried, ripe fruit of a low scrubby palm associated with prostate benefits.
- This botanical, composed of flavonoids, water-soluble polysaccharides, and free fatty acids, is believed to function as an anti-androgen and anti-inflammatory agent [258].
- Saw palmetto may reduce the growth of normal or malignant prostate cells by slowing the conversion of testosterone to DHT [259].
- Research also indicates beneficial effects on urinary flow rates in men with enlarged prostates, or benign prostatic hyperplasia (BPH) [258].
 - 320 mg daily seems to be effective.
- There is currently no conclusive scientific evidence regarding saw palmetto and prostate cancer.
- It has been theorized by some physicians that saw palmetto may, like the drug Proscar, lower PSA values. It is not clear, however, if there is any effect on the tumor. In other words, saw palmetto may mask the cancer.
- The use of saw palmetto may exclude an individual from participating in certain clinical trials.
- It is suggested to discontinue the use of saw palmetto 3 weeks prior to PSA testing.

TURMERIC (CURCUMIN)

- Curcumin, the yellow pigment and active component of turmeric, is a potent antioxidant*, which exhibits chemopreventive and growth inhibitory activity in several tumor cell lines [260-264].

- Evidence suggests that curcumin may suppress tumor initiation, promotion, and metastasis [260, 263, 265].
- Results indicate that curcumin is a novel and potent inducer of apoptosis* in both androgen-dependent and androgen-independent prostate cancer cells [260-261, 263-264].
- Additionally, curcumin promotes detoxification in the liver and possesses anti-inflammatory activity, possibly by inhibiting COX-2 activity [266].
- A unique anti-inflammatory herbal preparation (Zyflamend), which contains tumeric, has been found to inhibit both COX-1 and COX-2 activity, suppress cell growth, and induce apoptosis* in prostate cancer cells [267].

Hormone Therapy

- Patients who undergo hormone therapy may experience various side effects, notably hot flashes, night sweats, weight gain, and possibly a decline in bone mineral density.
- While the pathophysiology of hot flashes is complex, it is not yet well understood [268].
- Many studies have found that dietary and supplemental strategies do not appear to significantly affect hot flashes when compared to a placebo in women.
 - It should be noted, however, that the placebo effect has been considerable in various studies.
 - Nonetheless, some studies have found some benefit with soy isoflavones, black cohosh, red clover, and vitamin E [269-271].
 - Studies in men are lacking, but the results would not be expected to vary considerably.
- Similarly to women in menopause, men often report increased fat mass accompanied with a decrease in lean body mass while on androgen deprivation therapy (ADT) [272-276].
 - This change is concerning considering the evidence regarding increased body mass and prostate cancer risk.
- Multiple prospective studies have examined the relationship between ADT for prostate cancer and bone mineral density (BMD).
- Collectively, these studies suggest that significant bone loss is clearly observed within the first year of ADT [274, 277-282].
- Clinical trials are needed to evaluate the role of vitamin D, calcitonin, and oral bisphosphonates for the prevention and treatment of osteoporosis in patients undergoing ADT [275].
 - Trials are currently in process to evaluate the role of intravenous bisphosphonates.
- Nonetheless, dual-energy x-ray absorptiometry (DEXA) screening for osteoporosis is recommended prior to ADT, again at 1 year, and then at appropriate intervals thereafter, which may be every 2 years.
- Additionally, serum vitamin D (25-OH; 1,25-OH) should be assessed with these patients.
- Patients should be encouraged to maintain adequate yet not excessive amounts of dietary calcium and vitamin D.

- Furthermore, exercise, particularly resistance training may reduce risk osteoporosis and increase muscle mass [275].

Summary – Healthy Diet for Prostate Cancer

- Eat 8-10 colorful fruit and vegetable servings daily
 - 2-3 pieces of fruit
 - 1 cup or more of vegetables with lunch and dinner
 - 12 fl oz tomato-based juice
- Consume 30-35 grams of fiber daily
 - You will likely meet your fiber goal if you eat 8-10 servings of fruits and vegetables plus 1 serving beans/legumes or at least 2 servings of whole grains.
- Avoid processed and refined grains/flours/sugars.
 - Keep WHITE off your plate - bread, pasta, rice, cereal, cream sauces, cakes, and more.
- Limit meats and dairy
- Include healthy fats daily:
 - Examples include cold-water fish (i.e. salmon, trout, herring, sardines), flaxseed, walnuts, soybeans, avocados, and olive oil.
- Selenium (200 mcg)
 - 2 Brazil nuts or supplement
- Lycopene (30 mg)
 - ¾ C tomato sauce, 12 fl oz tomato juice, 4 ½ tbsp tomato paste
- Vitamin E (50-200 IU)
 - Natural vitamin E supplement that contains gamma-tocopherol
- Adequate vitamin D (400-2000 IU depending on age) to maintain serum 25-OH over 35 ng/mL.
- Drink 1-4 cups green tea daily
- Drink plenty of fluids, water or non-caffeinated beverages, daily to help meet fluid needs.
- Engage in daily physical activity to help achieve and maintain a healthy body weight.

PRACTICE PRECAUTION

- Always discuss changes in diet and supplement use with your physician.

WORDS OF WISDOM

“Let food be your medicine and medicine be your food.”
- Hippocrates

For additional information or resources, please visit the Ida and Joseph Friend Cancer Resource Center at 1600 Divisadero Street on the first floor, or call (415) 885-3693. The information in this publication is designed for educational purposes only and is not intended to replace the advice of your physician or health care provider, as each patient’s circumstances are individual. We encourage you to discuss with your physician any questions and concerns that you may have.

High-Fiber Sources

FRUITS:

Food	Serving Size	Fiber Grams/ Serving
Apple	1 medium	3.7
Banana	1 medium	2.8
Blackberries	½ cup	3.8
Blueberries	½ cup	1.9
Cantaloupe	1 cup	1.3
Figs (dried)	¼ cup	6.0
Grapefruit	1 medium	3.4
Grapes	1 cup	1.6
Guava	1 medium	4.9
Kiwi	1 medium	2.6
Orange	1 medium	3.1
Pear	1 medium	4.0
Persimmon	1 medium	6.0
Prunes	¼ cup	3.1

GRAINS & OTHER PRODUCTS:

Food	Serving Size	Fiber Grams/ Serving
Amaranth	¼ cup dry	7.4
Barley	½ cup cooked	3.0
Beans, black	½ cup cooked	8.3
Beans, red kidney	½ cup cooked	8.2
Beans, garbanzo	½ cup cooked	5.0
Bran cereals	¾ cup	Check labels (5.0-22.0)
Brown rice	½ cup cooked	1.4
Bulgur	½ cup cooked	4.0
Cream of wheat	½ cup cooked	0.5
Oatmeal	½ cup cooked	2.0
Peanuts	¼ cup	2.9
Quinoa	¼ cup dry	2.5
White rice	½ cup cooked	0.3

VEGETABLES:

Food	Serving Size	Fiber Grams/ Serving
Artichokes	1 medium	6.9
Beets	½ cup cooked	1.7
Broccoli	½ cup cooked	2.3
Brussels sprouts	½ cup cooked	2.0
Carrots	½ cup cooked	2.6
Kale	½ cup cooked	1.3
Lima beans	½ cup cooked	4.5
Peas, green	½ cup cooked	4.4
Spinach	½ cup cooked	2.2
Squash, winter-type	½ cup cooked	3.4
Sweet potatoes (yams)	½ cup cooked	2.7

Three Day Menu Plan: 3 Meals + Snack

This menu is based on 2100 calories, calories can be adjusted by altering portion sizes. The menu has been designed to serve as a guide in making healthy food choices. Experiment with substitutions as desired.

Day 1	Day 2	Day 3
Oatmeal (1 cup) Soy milk (1 cup) Flaxseed, ground (2 tbsp) Dried cranberries (1/4 cup) Brazil nuts (2 each)	Bagel, whole grain (1 each) Hummus (2 tbsp) Tomato (6 slices) Lemon pepper Cantaloupe (1 cup)	Tofu scramble Tofu (4 oz) Onions (1/4 cup) Peppers (1/2 cup) Mushrooms (1/2 cup) Toast, whole grain (2 slices) Jam (2 tbsp)
Turkey sandwich Whole grain bread (2 slices) Turkey (2 ½ oz) Lettuce (1 cup) Tomato (4 slices) Red peppers (1/4 cup) Onions (1/4 cup) Mustard (1 tsp) Peach (1 med)	Chunky Tortilla Soup (2 cups) Corn tortillas (2 each) Black beans (1/2 cup) Green salad (2 cups) Oil/vinegar dressing (2 tbsp)	Salad Spinach (2 cups) Broccoli (1/3 cup) Carrots (1/3 cup) Tomato (1/3 cup) Garbanzo beans (1/2 cup) Barley (1/2 cup) Walnuts (2 tbsp) Avocado (4 slices) Oil/vinegar dressing (2 tbsp) Whole-grain roll Orange (1 med)

Vegetable juice (12 oz) Granola bar (1 each)	Fruit smoothie Banana (1 each) Berries (1 cup) Flaxseed, ground (2 tbsp) Soy milk (1 cup) Brazil nuts (2 each)	Vegetable juice (12 oz) Popcorn (3 cups) Raw vegetables (1 cup) Hummus (1/4 cup)
Pasta, whole grain (2 cups) Tomato sauce (1 cup) Mushrooms (3/4 cup) Broccoli (1 cup) Green salad (2 cups) Light dressing (2 tbsp) Mixed fruit (1 cup)	Chicken & vegetable stir-fry Chicken breast (4 oz) Mixed vegetables (2 cups) Walnuts (2 tbsp) Brown rice (2 cups)	Salmon (4 oz) Quinoa (1 cup) with Brazil nuts (2 Tbsp) Asparagus (1 cup) Fruit salad (1 cup)

Recipes

Baked Tofu

Ingredients:

- 1 pound tofu, firm, drained
- 3-4 tbsp marinade or sauce (personal favorite: Veri Veri Teriyaki by Soy Vay)

Chop drained firm tofu into 1" cubes. Place tofu cubes in glass dish for baking. Pour marinade or sauce over tofu, stir well. Place tofu in oven at 350 F for 1 hour. Stir every 15-20 minutes.

Makes four 4-ounce servings.

Nutrition Information (per 4 oz serving):

Calories: 96	Dietary fiber: <1 gm
Protein: 8 gm	Sodium: 318 mg
Fat: 5 gm	Calcium: 155 mg (16% Daily Value)
Saturated fat: <1 gm	Iron: 1.4 mg (8% Daily Value)

Recipe developed by Natalie Ledesma, MS, RD

Washington Insider Salad

Ingredients:

- 1 can (15 oz) kidney beans, drained
- 1 can (15 oz) black eyed peas, drained
- 1 ½ cups cooked barley
- 6 tbsp cilantro, chopped finely
- 1 can (11 oz) corn
- 1 ½ cups tomatoes, diced
- 3 tbsp balsamic vinegar
- 2 tbsp olive oil

Prepare vegetables. Mix all ingredients together, and serve on a bed of dark green leafy lettuce. Add salt and pepper to taste.

Makes 8 servings (1 cup each).

Nutrition Information (per serving):

Calories: 215

Protein: 10 gm

Fat: 4 gm

Dietary fiber: 9 gm

Recipe developed by Sous Chef Chris at the Occidental Grill, Washington D.C.

Thick ‘N Chunky Tortilla Soup

Ingredients:

- 1 tbsp olive oil
- 1 cup chopped onions
- 2 garlic cloves, minced
- 3 medium sweet potatoes, cut into ½ ” cubes (4 cups)
- 3 cups chicken (or vegetable broth)
- ½ tsp cumin
- ½ tsp dried oregano leaves
- ½ tsp chili powder
- ¼ tsp ground red pepper (cayenne)
- 3 corn tortillas, cut into ½ ” strips
- 1 14.5-ounce can diced tomatoes, drained
- 1 cup corn (fresh, frozen, or canned)
- 1 4.5-ounce can chopped green chiles, undrained
- 2 tbsp fresh cilantro

Heat oven to 375 F. Heat oil in Dutch oven or 4-quart saucepan over medium heat until hot. Add onions and garlic; cook and stir 5-7 minutes or until onions are tender. Stir in sweet potatoes, broth, cumin, oregano, chili powder, and cayenne. Bring to a boil. Reduce heat to low; cover and simmer 10-15 minutes or until sweet potatoes are tender. Meanwhile, arrange tortilla strips in single layer on ungreased cookie sheet. Lightly spray strips with nonstick cooking spray. Bake at 375 F for 8-12 minutes or until golden brown and crisp. Cool and set aside. Transfer 2 ½ cups hot sweet potato mixture to food processor or blender; process until smooth. Return mixture to saucepan. Stir in tomatoes, corn, chiles, and cilantro. Cook over medium heat for 5 minutes or until thoroughly heated, stirring occasionally. To serve, spoon soup into individual soup bowls. Top each with crisp tortilla strips.

Makes 5 servings (1 ½ cups each).

Nutrition Information (per serving):

Calories: 280

Protein: 8 gm

Carbohydrate: 50 gm

Dietary fiber: 7 gm

Fat: 6 gm

Adapted from The Best of Italian, Mexican, & Chinese Cooking. Pillsbury Classic Cookbooks; January 1999.

Barbecued Tempeh with Bell Peppers

Ingredients:

- 8 ounce package tempeh
- 1 cup sliced onion
- 2 medium red or green bell peppers; sliced in strips
- 1 tsp minced garlic
- $\frac{3}{4}$ cup water
- $\frac{1}{4}$ cup tomato paste
- 1-2 tbsp molasses
- 1-2 tbsp brown sugar
- 2 tsp yellow mustard
- 2 tsp apple cider vinegar
- 1 tsp chili powder

Marinade

- $\frac{1}{2}$ cup tamari or soy sauce, low-sodium if desired
- 2 tbsp rice wine vinegar
- 4 tsp lemon juice
- 2 tsp honey

In a small bowl, combine marinade ingredients and mix well. Place tempeh in a shallow bowl and pour marinade over it. Cover and refrigerate several hours, turning occasionally. Drain tempeh and reserve marinade. Cut tempeh into $\frac{1}{2}$ " cubes. Coat bottom of large nonstick skillet with cooking spray and heat over medium heat until hot. Add onion and bell peppers and cook, stirring often, until just tender, about 5 minutes. Add marinade and all remaining ingredients. Bring mixture to a boil. Reduce heat and simmer, uncovered, until mixture thickens.

Makes 4 servings.

Nutrition Information (per serving):

Calories: 222

Protein: 18 gm

Fat: 5 gm

Saturated fat: 1 gm

Carbohydrate: 30 gm

Dietary Fiber: 7 gm

Recipe from Vegetarian Times, September 1998.

Spinach Spread

Ingredients:

- 1 package (10.5 ounces) silken tofu
- 1 tbsp lemon juice
- $\frac{1}{4}$ tsp garlic powder
- $\frac{3}{4}$ tsp onion powder
- $\frac{1}{2}$ tsp dried tarragon
- $\frac{1}{4}$ tsp salt
- 1 box (10 ounce) frozen chopped spinach, thawed
- 1 cup coarsely shredded carrots
- $\frac{1}{4}$ cup chopped green onion

Puree the tofu and lemon juice in blender until smooth. Whirl in the garlic and onion powders, tarragon, and salt just to blend. Scrape into a mixing bowl. Squeeze the spinach as dry as possible. Stir it into

the tofu, along with the carrots and green onion. Mix well. Serve with crackers, pita triangles, or vegetables.

Makes 8 servings (1/4 cup each).

Nutrition information (per serving):

Calories: 39	Sodium: 82 mg
Fat: 1 gm	Calcium: 51 mg
Saturated fat: 0 gm	Carbohydrate: 5 gm
Protein: 4 gm	Dietary Fiber: 2 gm

Recipe from the U.S. Soyfoods Directory, 1998.

Tofuntastico – Tofu Sauce

Ingredients:

- 1 package (12.3 ounce) silken tofu
- 1/2 cup water
- 3/4 cup fresh basil, chopped
- 4 tbsp nutritional yeast
- 3 tbsp Bragg's liquid aminos (or tamari or soy sauce)
- 1 tbsp lemon juice
- 1 tsp garlic, minced
- 3/4 tsp black pepper
- Alternative: Use lime/cilantro rather than lemon/basil

Blend all ingredients together in a blender or food processor. Serve over pasta, vegetables, baked potato, or other.

Makes 6 servings (1/2 cup each).

Nutrition Information (per serving):

Calories: 47	Carbohydrate: 4 gm
Protein: 7 gm	Dietary fiber: 2 gm
Fat: <1 gm	

Recipe developed by Natalie Ledesma, MS, RD

Alaska Salmon Bake with Walnut Crunch Coating

Ingredients:

- 1 pound salmon fillets, thawed if necessary
- 2 tbsp Dijon-style mustard
- 1-2 tbsp olive oil
- 4 tsp honey
- 1/4 cup bread crumbs
- 1/4 cup walnuts, finely chopped
- 2 tsp parsley, chopped
- Salt and pepper to taste
- Lemon wedges

Mix together mustard, olive oil, and honey in a small bowl; set aside. Mix together bread crumbs, walnuts, and parsley in a small bowl; set aside. Season each salmon fillet with salt and pepper. Place on a lightly greased baking sheet or broiling pan. Brush each fillet with mustard-honey mixture. Pat top of each fillet with bread crumb mixture. Bake at 450 F for 10 minutes per inch of thickness or

until salmon just flakes when tested with a fork. Serve with lemon wedges.
Makes 4 servings (4 oz each).

Nutrition Information (per serving):

Calories: 228

Protein: 20 gm

Fat: 12 gm

Omega-3 fatty acids: 1.7 gm

Adapted from Alaska Seafood Marketing Institute.

Vegan French Toast

Ingredients:

- 2 cups sliced bananas
- ¾ cup vanilla soy milk
- 1 tsp cinnamon, or ¾ tsp cinnamon plus ¼ tsp cardamom
- 8 sliced day-old whole grain bread
- 1 tsp canola oil
- 1 mango, peeled and cut

Place bananas, soy milk, cinnamon, and cardamom (if using) in a blender or food processor; blend until smooth. Pour mixture into a pie plate. Dip bread into mixture, turning to coat both sides. Scrape off excess batter. Brush a nonstick skillet or griddle lightly with oil or spray. When hot, add coated bread slices. Brown on one side, 2-3 minutes. Turn; brown other side. Transfer to plates; serve immediately with mango puree.

Makes 4 servings (2 slices each with 2 tbsp puree).

Nutrition Information (per serving):

Calories: 116

Carbohydrate: 23 gm

Protein: 4 gm

Dietary fiber: 3 gm

Fat: 2gm

Sodium: 137 mg

Recipe from Vegetarian Times, December 1995.

Banana Bread

Ingredients:

- ¾ cup ground flax seed
- 1 cup mashed banana
- ¼ cup apple juice concentrate
- ½ cup brown sugar
- ¼ cup applesauce
- Egg replacer for 2 eggs or 2 eggs (Ener-G Egg Replacer is made from potato starch & tapioca flour; works wonderfully in baked goods.)
- 1 ½ cup whole wheat pastry flour
- 1 tsp baking soda
- ½ tsp salt

**Additional optional ingredients may include ½ cup walnuts, raisins, or chocolate chips.

Mix all ingredients together. Pour in a coated 8"x4" pan. Bake at 350 F for about 40-45 minutes.

Makes 10 servings.

Nutrition Information (per serving):

Calories: 168 Carbohydrate: 29 gm
Protein: 5 gm Dietary fiber: 5 gm
Fat: 4 gm Omega-3 fatty acids: 1.4 gm

Recipe developed by Natalie Ledesma, MS, RD

Dilled Salmon Salad with Peas***Ingredients:***

- 1 can (15 oz) salmon, drained
- 1 package (16 oz) frozen peas, thawed
- ¼ cup lemon juice
- ¼ cup fresh dill (or 1-2 tbsp dried dill)
- 2 tbsp Dijon-style mustard
- 2 shallots, sliced thinly (about ½ cup)
- 1 bunch radishes (about 11 medium), thinly sliced
- 6 cups red leaf lettuce
- Salt and pepper to taste

Drain salmon, place in a mixing bowl, and break into pieces. Prepare the lemon juice, shallots, radishes, and lettuce. Add to the salmon the peas, lemon juice, dill, mustard, shallots, and radishes. Mix together gently. Add salt and pepper to taste. Serve salmon mixture over lettuce.

Makes 6 servings (2 cups each).

Nutrition Information (per serving):

Calories: 160
Protein: 17 gm
Fat: 4 gm
Dietary fiber: 5 gm

Adapted from the Women's Healthy Eating & Living Study (WHEL) at the University of California, San Diego. Developed by Vicky Newman, MS, RD, WHEL nutrition coordinator.

Neat Loaf***Ingredients:***

- 2 cups cooked brown rice
- 1 cup walnuts, finely chopped
- 1 onion, finely chopped
- ½ medium bell pepper, finely chopped
- 2 medium carrots, shredded or finely chopped
- 1 cup wheat germ
- 1 cup quick-cooking rolled oats
- ½ tsp each: thyme, marjoram, sage
- 2 tbsp soy sauce
- 2 tbsp stone ground or Dijon mustard
- Barbecue sauce or ketchup

Preheat the oven to 350 F. Combine all the ingredients except the barbecue sauce or ketchup. Mix for 2 minutes with a large spoon. This will help bind it together. Pat into an oil-sprayed 5x9" loaf pan and top with barbecue sauce or ketchup. Bake for 60 minutes. Let stand 10 minutes before serving.

Makes 8-10 servings.

Nutrition Information (per serving):

Calories: 204 Sodium: 248 mg
Protein: 9 gm Cholesterol: 0 mg
Fat: 9 gm
Carbohydrate: 19 gm

Recipe from The Peaceful Palate written by Jennifer Raymond, 1996.

Nutrition Resources

Books

The ABC's of Nutrition & Supplements and Prostate Cancer – written by Mark Moyad (2000)

Eating Your Way to Better Health: The Prostate Forum Nutrition Guide – written by Charles E. Myers Jr., M.D., Sara Sgarlat Steck, R.T. and Rose Sgarlat Myers, PT., Ph.D. (2000)

Food for Life: How the New Four Food Groups Can Save Your Life – written by Neal Barnard, M.D. (1994)

How to Prevent & Treat Cancer with Natural Medicine – written by Michael Murray, N.D. (2002)

The Color Code – written by James Joseph, Ph.D., Daniel Nadeau, M.D., & Anne Underwood (2002)

The Prostate Cancer Protection Plan – written by Robert Arnot, M.D. (2000)

Cookbooks

Cancer Lifeline Cookbook: Good Nutrition, Recipes, and Resources to Optimize the Lives of People Living with Cancer – written by Kimberly Mathai & Ginny Smith (2004)

Fat-Free and Easy: Great Meals in Minutes – written by Jennifer Raymond (vegetarian cookbook) (1997)

Lickety-Split Meals – written by Zonya Foco, R.D. (1998)

One Bite at a Time: Nourishing Recipes for People with Cancer, Survivors, & Their Caregivers – written by Rebecca Katz, Marsha Tomassi, & Mat Edelson (2004)

The Peaceful Palate – written by Jennifer Raymond (vegetarian cookbook) (1996)

12 Best Foods Cookbook: Over 200 Recipes Featuring the 12 Healthiest Foods - written by Dana Jacobi (2005)

Eat to Beat Prostate Cancer Cookbook – written by David Ricketts (2006)

Newsletters/Magazines

Cooking Light – www.cookinglight.com

Environmental Nutrition – <http://www.environmentalnutrition.com> (800) 829-5384

Nutrition Action Health Letter – <http://www.cspinet.org/nah/> Fax: (202) 265-4954

Websites

American Cancer Society – <http://www.cancer.org> (415) 394-7100

American Institute for Cancer Research – <http://www.aicr.org> (800) 843-8114

Cancer Nutrition Info (Provides up-to-date & comprehensive information on the connection between nutrition & cancer)
– www.cancernutritioninfo.com

CaP CURE - Good source of current research and articles – www.capcure.org

Center for Informed Food Choices – Offer cooking classes in the Bay Area that emphasize plant-based foods.
– www.informedeating.org

Consumer Lab – Evaluates quality of over-the-counter supplements <http://www.consumerlab.com>

Doctor's Guide to Prostate Cancer – www.pslgroup.com/prostcancer.htm

EarthSave International – Promotes food choices that are healthy for people & the planet.
– www.earthsave.org

Ida & Joseph Friend Cancer Resource Center – UCSF Mt.Zion – <http://cancer.ucsf.edu/crc> (415) 885-3693

National Cancer Institute <http://www.nci.nih.gov/> (800) 4-CANCER (800-422-6237)

Oncolink – Provides information regarding clinical trials, newsgroups, psychosocial support, & more.
– <http://oncolink.upenn.edu>

Physicians Committee for Responsible Medicine – Nonprofit organization that promotes preventive medicine, conducts clinical research, and encourages higher standards for ethics & effectiveness in research.
– <http://www.pcrm.org/index.html>

Prostate Pointers - Comprehensive prostate cancer web site with a full range of subjects pertinent to prostate cancer – www.prostatepointers.com

San Francisco Vegetarian Society – Monthly restaurant outings & pot-luck dinners; call 415-273-5481.
– <http://www.sfvs.org>

US TOO International, Inc. - Nonprofit organization offers descriptions of treatments, online newsletter, glossary of medical terms & information on local support groups – www.ustoo.com

The Vegetarian Resource Group - Provides vegetarian nutrition information & vegetarian recipes
– www.vrg.org

WebMD – <http://my.webmd.com>

Glossary

Angiogenesis – The formation of new blood vessels.

Antioxidant – A substance that inhibits oxidation or inhibits reactions promoted by oxygen or peroxides.

Apoptosis – Programmed cell death.

Carcinogenesis – Beginning of cancer development.

Case-Control Studies – An epidemiological study in which a group of, say, cancer patients (cases) is compared to a similar but cancer-free population (controls) to help establish wheth-

er the past or recent history of a specific exposure such as smoking, alcohol consumption and dietary intake, etc. are causally related the risk of disease.

Catechin – One of the tannic acids; phytonutrient, specifically, one of the flavonoids found in green tea.

Creatine – An amino acid that is formed in the muscle tissue of vertebrates; supplies energy for muscle contraction.

Cohort Studies – Follow-up study of a (usually large) group of people, initially disease-free. Differences in disease incidence within the cohort are calculated in relation to different levels of exposure to specific factors, such as smoking, alcohol consumption, diet and exercise, that were measured at the start of the study and, sometimes, at later times during the study.

Eicosanoids – Biologically active compounds that regulate blood pressure, blood clotting, and other body functions. They include prostaglandins, thromboxanes, and leukotrienes.

Endogenous – Originating from within, as within the body.

Epidemiological – Of or relating to epidemiology; the branch of medicine that involved the study of the causes, distribution, and control of disease in populations.

Glutathione – A polypeptide produced primarily in the liver; involved in DNA synthesis and repair, protein and prostaglandin synthesis, amino acid transport, metabolism of toxins and carcinogens, immune system function, prevention of oxidative cell damage, and enzyme activation.

Insulin – Insulin is a hormone produced by the pancreas in the body that regulates the metabolism of carbohydrates and fats, especially the conversion of glucose to glycogen, which lowers the body's blood sugar level.

Lignans – Phytoestrogens that have a similar chemical structure to estradiol and tamoxifen; appear to offer protection against breast cancer.

Meta-analysis – The process of using statistical methods to combine the results of different studies.

Mutation – Abnormal cell development.

Nitrosamines – Derivatives of nitrites that may be formed in the stomach when nitrites combine with amines; carcinogenic in animals.

Phytonutrients – Plant compounds that appear to have health-protecting properties.

Polyphenols – Phytonutrients that act as an antioxidant; compounds that protects the cells and body chemicals against damage caused by free radicals, reactive atoms that contribute to tissue damage in the body.

Sex hormone – binding globulin (SHBG) – A protein in the blood that acts as a carrier for androgens and estradiol; inhibits the estradiol-induced proliferation of breast cancer cells.

References

1. Heber, D., Fair, W., & Ornish, D. (1999). Nutrition & Prostate Cancer: A Monograph from the CaP CURE Nutrition Project. 2nd edition. January <http://www.capcure.org.il/abstracts/pub-pdf/nutrition.pdf>.
2. Byers, T., Nestle, M., McTiernan, A., Doyle, C., Currie-Williams, A., Gansler, T., et al. American Cancer Society 2001 Nutrition and Physical Activity Guidelines Advisory Committee. (2002). 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*, 52(2), 92-119.
3. Ornish, D., Weidner, G., Fair, W.R., Marlin, R., & Pettengill, E.B. (2005). Intensive lifestyle changes may affect the progression of prostate cancer. *Journal of Urology*, 174(3), 1065-1069.
4. Saxe, G.A., Major, J.M., Nguyen, J.Y., Freeman, K.M., & Downs, T.M. (2006). Potential attenuation of disease progression in recurrent prostate cancer with plant-based diet and stress reduction. *Integrative Cancer Therapies*, 5(3), 206-213.
5. Dubenmier, J.J., Weidner, G., Marlin, R., Crutchfield, L., & Dunn-Emke, S. (2006). Lifestyle and health-related quality of life of men with prostate cancer managed with active surveillance. *Urology*, 67(1), 125-130.
6. Sunny, L. (2005). A low fat diet rich in fruits and vegetables may reduce the risk of developing prostate cancer. *Asian Pacific Journal of Cancer Prevention*, 6(4), 490-496.
7. Khaw, K.T., Bingham, S., Welch, R.L.A., Wareham, N., Oakes, S., Day, N., et al. (2001). The European Prospective Investigation into Cancer and Nutrition. Relation between plasma ascorbic acid and mortality in men and women in EPIC-Norfolk prospective study: a prospective population study. *Lancet*, 357, 657-663.
8. Willett, W.C. (2000). Intakes of fruits, vegetables and related nutrients and the risk of non-Hodgkin's lymphoma among women. *Cancer Epidemiology Biomarkers and Prevention*, 9, 477-485.
9. U.S. Dept. of Agriculture. (1995). Nutrition and your health: Dietary Guidelines for Americans, ed. 4, Home & Garden Bulletin No. 232, Washington DC, U.S. Dept. of Health and Human Services.
10. World Cancer Research Fund/American Institute for Cancer Research. Food, nutrition and the prevention of cancer: A global perspective. Washington, DC: American Institute for Cancer Research, 1997.
11. Steinmetz, K.A., & Potter, J.D. (1996). Vegetables, fruit, and cancer prevention: a review. *Journal of American Dietetic Association*, 96, 1027-1039.
12. Zhang, S., Hunter, D.J., Forman, M.R., Rosner, B.A., Speizer, F.E., Colditz, G.A., et al. (1999). Dietary carotenoids and vitamins A, C, and E and risk of breast cancer. *Journal of the National Cancer Institute*, 91, 547-556.
13. Zhang, S.M., Hunter, D.J., Rosner, B.A., Giovannucci, E.L., Colditz, G.A., Speizer, F.E., et al. (2000). Intakes of fruits, vegetables, and related nutrients and the risk of non-Hodgkin's lymphoma among women. *Cancer Epidemiology Biomarkers and Prevention*, 9(5), 477-485.
14. Freudenheim, J.L., Marshall, J.R., Vena, J.E., Laughlin, R., Brasure, J.R., Swanson, M.K., et al. (1996). Premenopausal breast cancer risk and intake of vegetables, fruits, and related nutrients. *Journal of the National Cancer Institute*, 88, 340-348.
15. Rock, Cl., Saxe, G.A., Ruffin, M.T. 4th, August, D.A., & Schottenfeld, D. (1996). Carotenoids, vitamin A, and estrogen receptor status in breast cancer. *Nutrition and Cancer*, 25(3), 281-296.
16. Eastwood, M.A. (1999). Interaction of dietary antioxidants in vivo: How fruit and vegetables prevent disease? *Quarterly Journal of Medicine*, (92), 527-530.
17. Voorrips, L.E., Goldbohm, R.A., Brants, H.A., van Poppel, G.A., Sturmans, F., Hermus, R.J., et al. (2000). A prospective cohort study on antioxidant and folate intake and male lung cancer risk. *Cancer Epidemiology Biomarkers and Prevention*, 9, 357-365.
18. Nguyen, J.Y., Major, J.M., Knott, C.J., Freeman, K.M., & Downs, T.M. (2006). Adoption of a plant-based diet by patients with recurrent prostate cancer. *Integrative Cancer Therapies*, 5(3), 214-223.
19. Hodge, A.M., English, D.R., McCredie, M.R., Severi, G., Boyle, P., Hopper, J.L., et al. (2004). Foods, nutrients and prostate cancer. *Cancer Causes Control*, 15(1), 11-20.
20. Cohen, J.H., Kristal, A.R., & Stanford, J.L. (2000). Fruit and vegetable intakes and prostate cancer risk. *Journal of the National Cancer Institute*, 92(1), 61-68.
21. Jain, M.G., Hislop, G.T., Howe, G.R., & Ghadirian, P. (1999). Plant foods, antioxidants, and prostate cancer risk: findings from case-control studies in Canada. *Nutrition and Cancer*, 34(2), 173-184.
22. Deneo-Pellegrini, H., De Stefani, E., Ronco, A., & Mendilaharsu, M. (1999). Foods, nutrients and prostate cancer: a case-control study in Uruguay. *British Journal of Cancer*, 80(3-4), 591-597.
23. Wang, L., Liu, D., Ahmed, T., Chung, F.L., Conaway, C., Chiao, J.W., et al. (2004). Targeting cell cycle machinery as a molecular mechanism of sulforaphane in prostate cancer prevention. *International Journal of Oncology*, 24(1), 187-192.
24. Kolonel, L.N., Hankin, J.H., Whittemore, A.S., Wu, A.H., Gallagher, R.P., Wilkens, L.R., et al. (2000). Vegetables, fruits, legumes and prostate cancer: a multiethnic case-control study. *Cancer Epidemiology Biomarkers and Prevention*, 9(8), 795-804.
25. Chinni, S.R., Li, Y., Upadhyay, S., Koppolu, P.K., Sarkar, F.H. (2001). Indole-3-carbinol (I3C) induced cell growth inhibition, G1 cell cycle arrest and apoptosis in prostate cancer cells. *Oncogene*, 20(23), 2927-2936.
26. Canene-Adams, K., Lindshield, B.L., Wang, S., Jeffrey, E.H., Clinton, S.K., et al. (2007). Combinations of Tomato and Broccoli Enhance Antitumor Activity in Dunning R3327-H Prostate Adenocarcinomas. *Cancer Research*, 67(2), 836-843.
27. Garikapaty, V.P., Ashok, B.T., Tadi, K., Mittelman, A., & Tiwari, R.K. (2006). 3,3'-Diindolylmethane downregulates pro-survival pathway in hormone independent prostate cancer. *Biochemical and Biophysical Research Communications*, 340(2), 718-725.
28. Bosetti, C., Tzonou, A., Lagiou, P., Negri, E., Trichopoulos, D., & Hsieh, C.C., et al. (2000). Fraction of prostate cancer incidence attributed to diet in Athens, Greece. *European Journal of Cancer Prevention*, 9(2), 119-123.
29. Norrish, A.E., Jackson, R.T., Sharpe, S.J., & Skeaff, C.M. (2000). Prostate cancer and dietary carotenoids. *American Journal of Epidemiology*, 151(2), 119-123.
30. Mills, P.K., Beeson, W.L., Phillips, R.L., & Fraser, G.E. (1989). Cohort study of diet, lifestyle, and prostate cancer in Adventist men. *Cancer*, 64(3), 598-604.
31. Tzonou, A., Signorello, L.B., Lagiou, P., Wu, J., Trichopoulos, D., & Trichopoulou, A. (1999). Diet and cancer of the prostate: a case-control study in Greece. *International Journal of Cancer*, 80(5), 704-708.
32. Giovannucci, E., Ascherio, A., Rimm, E.B., Stampfer, M.J., Colditz, G.A., Willett, W.C., et al. (1995). Intake of carotenoids and retinol in relation to risk of prostate cancer. *Journal of the National Cancer Institute*, 87(23), 1767-1776.

33. Wu, K., Erdman, J.W. Jr, Schwartz, S.J., Platz, E.A., Leitzmann, M., Clinton, S.K., et al. (2004). Plasma and Dietary Carotenoids, and the Risk of Prostate Cancer: A Nested Case-Control Study. *Cancer Epidemiology Biomarkers and Prevention*, 13(2), 260-269.
34. Key, T.J., Silcocks, P.B., Davey, G.K., Appleby, P.N., & Bishop, D.T. (1997). A case-control study of diet and prostate cancer. *British Journal of Cancer*, 76(5), 678-687.
35. Ohno, Y., Yoshida, O., Oishi, K., Okada, K., Yamabe, H., Schroeder, F.H., et al. (1998). Dietary beta-carotene and cancer of the prostate: a case-control study in Kyoto, Japan. *Cancer Research*, 48(5), 1331-1336.
36. Pierce, J.P., Faerber, S., Wright, F.A., Rock, C.L., Newman, V., Flatt, S.W., et al. (2002). A randomized trial of the effect of a plant-based dietary pattern on additional breast cancer events and survival: the Women's Healthy Eating and Living (WHEL) Study. *Controlled Clinical Trials*, 23(6), 728-756.
37. Lombardi-Boccia, G., Lucarini, M., Lanzi, S., Aguzzi, A., & Cappelloni, M. (2004). Nutrients and antioxidant molecules in yellow plums (*Prunus domestica* L.) from conventional and organic productions: a comparative study. *Journal of Agricultural Food Chemistry*, 52(1), 90-94.
38. Grønder-Pedersen, L., Rasmussen, S.E., Bugel, S., Jørgensen, L.V., Dragsted, L.O., et al. (2003). Effect of diets based on foods from conventional versus organic production on intake and excretion of flavonoids and markers of antioxidative defense in humans. *Journal of Agricultural Food Chemistry*, 51(19), 5671-5676.
39. Asami, D.K., Hong, Y.J., Barrett, D.M., & Mitchell, A.E. (2003). Comparison of the total phenolic and ascorbic acid content of freeze-dried and air-dried marionberry, strawberry, and corn grown using conventional, organic, and sustainable agricultural practices. *Journal of Agricultural Food Chemistry*, 51(5), 1237-1241.
40. Baxter, G.J., Graham, A.B., Lawrence, J.R., Wiles, D., & Paterson, J.R. (2001). Salicylic acid in soups prepared from organically and non-organically grown vegetables. *European Journal of Nutrition*, 40(6), 289-292.
41. Ferreres, F., Valentao, P., Llorach, R., Pinheiro, C., Cardoso, L., Pereira, J.A., et al. (2005). Phenolic compounds in external leaves of tronchuda cabbage (*Brassica oleracea* L. var. costata DC). *Journal of Agricultural Food Chemistry*, 53(8), 2901-2907.
42. Harris, P.J., Robertson, A.M., Watson, M.E., Triggs, C.M., & Ferguson, L.R. (1993). The effects of soluble-fiber polysaccharides on the adsorption of a hydrophobic carcinogen to an insoluble dietary fiber. *Nutrition and Cancer*, 19(1), 43-54.
43. Slavin, J.L. (2000). Mechanisms for the impact of whole grain foods on cancer risk. *Journal of the American College of Nutrition*, 19(3 Suppl), 300S-307S.
44. Slavin, J. (2003). Why whole grains are protective: biological mechanisms. *The Proceedings Nutrition Society*, 62(1), 129-134.
45. Tariq, N., Jenkins, D.J., Vidgen, E., Fleshner, N., Kendall, C.W., Story, J.A., et al. (2000). Effect of soluble and insoluble fiber diets on serum prostate specific antigen in men. *Journal of Urology*, 163(1), 114-118.
46. Tymchuk, C.N., Barnard, R.J., Heber, D., & Aronson, W.J. (2001). Evidence of an inhibitory effect of diet and exercise on prostate cancer cell growth. *The Journal of Urology*, 166(3), 1185-1189.
47. Pelucchi, C., Talamini, R., Galeone, C., Negri, E., Franceschi, S., Dal Maso, L., et al. (2004). Fibre intake and prostate cancer risk. *International Journal of Cancer*, 109(2), 278-280.
48. La Vecchia, C., Chatenoud, L., Negri, E., & Franceschi, S. (2003). Session: whole cereal grains, fibre and human cancer wholegrain cereals and cancer in Italy. *The Proceedings Nutrition Society*, 62(1), 45-49.
49. Shike, M., Latkany, L., Riedel, E., Fleisher, M., Schatzkin, A., Lanza, E., et al. (2002). Lack of effect of a low-fat, high-fruit, -vegetable, and -fiber diet on serum prostate-specific antigen of men without prostate cancer: results from a randomized trial. *Journal of Clinical Oncology*, 20(17), 3592-3598.
50. Hebert, J.R., Hurley, T.G., Olendzki, B.C., Teas, J., Ma, Y., Hampl, J.S., et al. (1998). Nutritional and socioeconomic factors in relation to prostate cancer mortality: a cross-national study. *Journal of the National Cancer Institute*, 90, 1637-1647.
51. Stoll, B.A. (1996). Can supplementary dietary fibre suppress breast cancer growth? *British Journal of Cancer*, 73(5), 557-559.
52. Williams, G.M., Williams, C.L., & Weisburger, J.H. (1999). Diet and cancer prevention: the fiber first diet. *Toxicological Sciences*, 52(2 Suppl), 72-86.
53. Hsing, A.W., Chua, S. Jr, Gao, Y.T., Gentszsch, E., Chang, L., Deng, J., et al. (2001). Prostate cancer risk and serum levels of insulin and leptin: a population-based study. *Journal of the National Cancer Institute*, 93(10), 783-789.
54. Barnard, R.J., Ngo, T.H., Leung, P.S., Aronson, W.J., & Golding, L.A. (2003). A low-fat diet and/or strenuous exercise alters the IGF axis in vivo and reduces prostate tumor cell growth in vitro. *Prostate*, 56(3), 201-206.
55. Ngo, T.H., Barnard, R.J., Tymchuk, C.N., Cohen, P., & Aronson, W.J. (2002). Effect of diet and exercise on serum insulin, IGF-I, and IGF-BP-1 levels and growth of LNCaP cells in vitro (United States). *Cancer Causes Control*, 13(10), 929-935.
56. Moyad, M.A. (2003). The use of complementary/preventive medicine to prevent prostate cancer recurrence/progression following definitive therapy: part I--lifestyle changes. *Current Opinion in Urology*, 13(2), 137-145.
57. Aksoy, Y., Aksoy, H., Bakan, E., Atmaca, A.F., & Akcay, F. (2004). Serum insulin-like growth factor-I and insulin-like growth factor-binding protein-3 in localized, metastasized prostate cancer and benign prostatic hyperplasia. *Urologia Internationalis*, 72(1), 62-65.
58. Yu, H., & Berkel, H. (1999). Insulin-like growth factors and cancer. *Journal of the Louisiana State Medical Society*, 151(4), 218-223.
59. Li, L., Yu, H., Schumacher, F., Casey, G., & Witte, J.S. (2003). Relation of serum insulin-like growth factor-I (IGF-I) and IGF binding protein-3 to risk of prostate cancer (United States). *Cancer Causes Control*, 14(8), 721-726.
60. Cardillo, M.R., Monti, S., Di Silverio, F., Gentile, V., Sciarra, F., Toscano, V., et al. (2003). Insulin-like growth factor (IGF)-I, IGF-II and IGF type I receptor (IGFR-I) expression in prostatic cancer. *Anticancer Research*, 23(5A), 3825-3835.
61. Kaaks, R., & Lukanova, A. (2001). Energy balance and cancer: the role of insulin and insulin-like growth factor-I. *Proceedings of the Nutrition Society*, 60(1), 91-106.
62. Spentzos, D., Mantzoros, C., Regan, M.M., Morrissey, M.E., Duggan, S., Flickner-Garvey, S., et al. (2003). Minimal effect of a low-fat/high soy diet for asymptomatic, hormonally naive prostate cancer patients. *Clinical Cancer Research*, 9(9), 3282-3287.
63. Habito, R.C., & Ball, M.J. (2001). Postprandial changes in sex hormones after meals of different composition. *Metabolism*, 50(5), 505-511.
64. Fleshner, N., Bagnell, P.S., Klotz, L., & Venkateswaran, V. (2004). Dietary fat and prostate cancer. *The Journal of Urology*, 171(2 Pt 2), S19-S24.
65. Veierød, M.B., Laake, P., & Thelle, D.S. (1997). Dietary fat intake and risk of prostate cancer: a prospective study of 25,708 Norwegian men. *International Journal of Cancer*, 73(5), 634-638.
66. Bairati, I., Meyer, F., Fradet, Y., & Moore, L. (1998). Dietary fat and advanced prostate cancer. *The Journal of Urology*, 159(4), 1271-1275.

67. Meyer, F., Bairati, I., Shadmani, R., Fradet, Y., & Moore, L. (1999). Dietary fat and prostate cancer survival. *Cancer Causes Control*, 10(4), 245-251.
68. Le Marchand, L., Hankin, J.H., Kolonel, L.N., & Wilkens, L.R. (1991). Vegetable and fruit consumption in relation to prostate cancer risk in Hawaii: a reevaluation of the effect of dietary beta-carotene. *American Journal of Epidemiology*, 133(3), 215-219.
69. Giovannucci, E., Rimm, E.B., Colditz, G.A., Stampfer, M.J., Ascherio, A., Chute, C.C., et al. (1993). A prospective study of dietary fat and risk of prostate cancer. *Journal of the National Cancer Institute*, 85(19), 1571-1579.
70. Colli, J.L., & Colli, A. (2005). Comparisons of prostate cancer mortality rates with dietary practices in the United States. *Urologic Oncology*, 23(6), 390-398.
71. Lee, M.M., Wang, R.T., Hsing, A.W., Gu, F.L., Wang, T., Spitz, M., et al. (1998). Case-control study of diet and prostate cancer in China. *Cancer Causes Control*, 9(6), 545-552.
72. Kushi, L., & Giovannucci, E. (2002). Dietary fat and cancer. *The American Journal of Medicine*, 113 Suppl 9B, 63S-70S.
73. Meyer, F., Bairati, I., Fradet, Y., & Moore, L. (1997). Dietary energy and nutrients in relation to preclinical prostate cancer. *Nutrition Cancer*, 29(2), 120-126.
74. Fradet, Y., Meyer, F., Bairati, I., Shadmani, R., & Moore, L. Dietary fat and prostate cancer progression and survival. *European Urology*, 35(5-6), 388-391.
75. Michaud, D.S., Augustsson, K., Rimm, E.B., Stampfer, M.J., Willett, W.C., Giovannucci, E., et al. (2001). A prospective study on intake of animal products and risk of prostate cancer. *Cancer Causes Control*, 12(6), 557-567.
76. Ramon, J.M., Bou, R., Romea, S., Alkiza, M.E., Jacas, M., Ribes, J., et al. (2000). Dietary fat intake and prostate cancer risk: a case-control study in Spain. *Cancer Causes Control*, 11(8), 679-685.
77. Lemaitre, R.N., King, I.B., & Raghunathan, T.E. (2002). Cell membrane trans-fatty acids and the risk of primary cardiac arrest. *Circulation*, 105, 697-701.
78. Katan, M.B. (2000). Trans fatty acids and plasma lipoproteins. *Nutrition Reviews*, 58, 188-191.
79. Mensink R.P., & Katan M.B. (1990). Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *New England Journal of Medicine*, 323, 439-445.
80. Nelson, G.J. (1998). Dietary fat, trans fatty acids, and risk of coronary heart disease. *Nutrition Reviews*, 56, 250-252.
81. Bakker, N., Van't, Veer, P., & Zock, P.L. (1997). Adipose fatty acids and cancers of the breast, prostate and colon: an ecological study. EURAMIC Study Group. *International Journal of Cancer*, 72(4), 587-591.
82. Slattery, M.L., Benson, J., Ma, K.N., Schaffer, D., & Potter, J.D. (2001). Trans-fatty acids and colon cancer. *Nutrition and Cancer*, 39(2), 170-175.
83. Hughes-Fulford, M., Chen, Y., & Tjandrawinata, R.R. (2001). Fatty acid regulates gene expression and growth of human prostate cancer PC-3 cells. *Carcinogenesis*, 22(5), 701-707.
84. Norrish, A.E., Jackson, R.T., Sharpe, S.J., & Skeaff, C.M. (2000). Men who consume vegetable oils rich in monounsaturated fat: their dietary patterns and risk of prostate cancer (New Zealand). *Cancer Causes Control*, 11(7), 609-615.
85. Gonzalez, C.A. & Salas-Salvado, J. (2006). The potential of nuts in the prevention of cancer. *British Journal of Nutrition*, 96 Suppl 2, S87-S94.
86. Lu, Q.Y., Arteaga, J.R., Zhang, Q., Huerta, S., Go, V.L., et al. (2005). Inhibition of prostate cancer cell growth by an avocado extract: role of lipid-soluble bioactive substances. *Journal of Nutritional Biochemistry*, 16(1), 23-30.
87. Mannisto, S., Pietinen, P., Virtanen, M.J., Salminen, I., Albanes, D., Giovannucci, E., Virtamo, J., et al. (2003). Fatty acids and risk of prostate cancer in a nested case-control study in male smokers. *Cancer Epidemiology Biomarkers and Prevention*, 12(12), 1422-1428.
88. Harvei, S., Bjerve, K.S., Tretli, S., Jellum, E., Robsahm, T.E., Vatten, L., et al. (1997). Prediagnostic level of fatty acids in serum phospholipids: omega-3 and omega-6 fatty acids and the risk of prostate cancer. *International Journal of Cancer*, 71(4), 545-551.
89. Gann, P.H., Hennekens, C.H., Sacks, F.M., Grodstein, F., Giovannucci, E.L., Stampfer, M.J., et al. (1994). Prospective study of plasma fatty acids and risk of prostate cancer. *Journal of the National Cancer Institute*, 86(4), 281-286.
90. Newcomer, L.M., King, I.B., Wicklund, K.G., & Stanford, J.L. (2001). The association of fatty acids with prostate cancer risk. *Prostate*, 47(4), 262-268.
91. Godley, P.A., Campbell, M.K., Gallagher, P., Martinson, F.E., Mohler, J.L., Sandler, R.S., et al. (1996). Biomarkers of essential fatty acid consumption and risk of prostatic carcinoma. *Cancer Epidemiology Biomarkers and Prevention*, 5(11), 889-895.
92. Ghosh, J., & Myers, C.E. (1997). Arachidonic acid stimulates prostate cancer cell growth: critical role of 5-lipoxygenase. *Biochemical Biophysical Research Communications*, 235(2), 418-423.
93. Ritch, C.R., Wan, R.L., Stephens, L.B., Taxy, J.B., Huo, D., Gong, E.M., et al. (2007). Dietary fatty acids correlate with prostate cancer biopsy grade and volume in Jamaican men. *Journal of Urology*, 177(1), 97-101.
94. Kelavkar, U.P., Hutzley, J., Dhir, R., Kim, P., Allen, K.G., et al. (2006). Prostate tumor growth and recurrence can be modulated by the omega-6:omega-3 ratio in diet: athymic mouse xenograft model simulating radical prostatectomy. *Neoplasia*, 8(2), 112-124.
95. Norrish, A.E., Skeaff, C.M., Arribas, G.L., Sharpe, S.J., & Jackson, R.T. (1999). Prostate cancer risk and consumption of fish oils: a dietary biomarker-based case-control study. *British Journal of Cancer*, 81(7), 1238-1242.
96. Augustsson, K., Michaud, D.S., Rimm, E.B., Leitzmann, M.F., Stampfer, M.J., Willett, W.C., et al. (2003). A prospective study of intake of fish and marine fatty acids and prostate cancer. *Cancer Epidemiology Biomarkers and Prevention*, 12(1), 64-67.
97. Terry, P., Lichtenstein, P., Feychting, M., Ahlbom, A., & Wolk, A. (2001). Fatty fish consumption and risk of prostate cancer. *Lancet*, 357(9270), 1764-1766.
98. Terry, P.D., Rohan, T.E., & Wolk, A. (2003). Intakes of fish and marine fatty acids and the risks of cancers of the breast and prostate and of other hormone-related cancers: a review of the epidemiologic evidence. *The American Journal of Clinical Nutrition*, 77(3), 532-543.
99. Chung, B.H., Mitchell, S.H., Zhang, J.S., & Young, C.Y. (2001). Effects of docosahexaenoic acid and eicosapentaenoic acid on androgen-mediated cell growth and gene expression in LNCaP prostate cancer cells. *Carcinogenesis*, 22(8), 1201-1206.
100. Kobayashi, N., Barnard, R.J., Henning, S.M., Flashoff, D., Reddy, S.T., et al. (2006). Effect of altering dietary omega-6/omega-3 fatty acid ratios on prostate cancer membrane composition, cyclooxygenase-2, and prostaglandin E2. *Clinical Cancer Research*, 12(15), 4662-4670.
101. Chan, J.M., Holick, C.N., Leitzmann, M.F., Rimm, E.B., Willett, W.C., et al. (2006). Diet after diagnosis and the risk of prostate cancer progression, recurrence, and death (United States). *Cancer Causes Control*, 17(2), 199-208.
102. Rose, D.P., & Connolly, J.M. (1991). Effects of fatty acids and eicosanoid synthesis inhibitors on the growth of two human prostate cancer cell lines. *Prostate*, 18(3), 243-254.

103. Harris, P.J., Robertson, A.M., Watson, M.E., Triggs, C.M., & Ferguson, L.R. (1993). The effects of soluble-fiber polysaccharides on the adsorption of a hydrophobic carcinogen to an insoluble dietary fiber. *Nutrition and Cancer*, 19(1), 43-54.
104. Davis, B.C., & Kris-Etherton, P.M. (2003). Achieving optimal essential fatty acid status in vegetarians: current knowledge and practical implications. *American Journal of Clinical Nutrition*, 78(3 Suppl), 640S-646S.
105. Gerster, H. (1998). Can adults adequately convert alpha-linolenic acid (18:3n-3) to eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3)? *International Journal for Vitamin and Nutrition Research*, 68(3), 159-173.
106. Andersson, S.O., Wolk, A., Bergstrom, R., Giovannucci, E., Lindgren, C., Baron, J., et al. (1996). Energy, nutrient intake and prostate cancer risk: a population-based case-control study in Sweden. *International Journal of Cancer*, 68(6), 716-722.
107. De Stefani, E., Deneo-Pellegrini, H., Boffetta, P., Ronco, A., & Mendilaharsu, M. (2000). Alpha-linolenic acid and risk of prostate cancer: a case-control study in Uruguay. *Cancer Epidemiology Biomarkers and Prevention*, 9(3), 335-338.
108. Rohan, T.E., Howe, G.R., Burch, J.D., & Jain, M. (1995). Dietary factors and risk of prostate cancer: a case-control study in Ontario, Canada. *Cancer Causes Control*, 6(2), 145-154.
109. Kristal, A.R., Cohen, J.H., Qu, P., & Stanford, J.L. (2002). Associations of energy, fat, calcium, and vitamin D with prostate cancer risk. *Cancer Epidemiology Biomarkers and Prevention*, 11(8), 719-725.
110. Hsieh, L.J., Carter, H.B., Landis, P.K., Tucker, K.L., Metter, E.J., Newschaffer, C.J., et al. (2003). Association of energy intake with prostate cancer in a long-term aging study: Baltimore Longitudinal Study of Aging (United States). *Urology*, 61(2), 297-301.
111. Sonntag, W.E., Lynch, C.D., Cefalu, W.T., Ingram, R.L., Bennett, S.A., Thornton, P.L., et al. (1999). Pleiotropic effects of growth hormone and insulin-like growth factor (IGF)-1 on biological aging: inferences from moderate caloric-restricted animals. *The Journal of Gerontology. Series A, Biological Science & Medical Science*, 54(12), B521-B538.
112. Calle. (2003). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *The New England Journal of Medicine*, 348, 1625-1638.
113. Amling, C.L., Riffenburgh, R.H., Sun, L., Moul, J.W., Lance, R.S., Kusuda, L., et al. (2004). Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. *Journal of Clinical Oncology*, 22(3), 439-445.
114. Freedland, S.J., Aronson, W.J., Kane, C.J., Presti, J.C. Jr, Amling, C.L., Elashoff, D., et al. (2004). Impact of obesity on biochemical control after radical prostatectomy for clinically localized prostate cancer: a report by the Shared Equal Access Regional Cancer Hospital database study group. *Journal of Clinical Oncology*, 22(3), 446-53.
115. Rodriguez, C., Freedland, S.J., Deka, A., Jacobs, E.J., McCullough, M.L., et al. (2007). Body Mass Index, Weight Change, and Risk of Prostate Cancer in the Cancer Prevention Study II Nutrition Cohort. *Cancer Epidemiology and Biomarkers Prevention*, 16(1), 63-69.
116. Bassett, W.W., Cooperberg, M.R., Sadetsky, N., Silva, S., DuChane, J., Pasta, D.J., et al. (2005). Impact of obesity on prostate cancer recurrence after radical prostatectomy: data from CaPSURE. *Urology*, 66(5), 1060-1065.
117. Nilsen, T.I., Romundstad, P.R., & Vatten, L.J. (2006). Recreational physical activity and risk of prostate cancer: A prospective population-based study in Norway (the HUNT study). *International Journal of Cancer*, 119(12), 2943-2947.
118. Ellinger, S., Ellinger, J., & Stehle, P. (2006). Tomatoes, tomato products and lycopene in the prevention and treatment of prostate cancer: do we have the evidence from intervention studies? *Current Opinion in Clinical Nutrition and Metabolic Care*, 9(6), 722-727.
119. Hwang, E.S., & Bowen, P.E. (2002). Can the consumption of tomatoes or lycopene reduce cancer risk? *Integrative Cancer Therapies*, 1(2), 121-132.
120. Lu, Q.Y., Hung, J.C., Heber, D., Go, V.L., Reuter, V.E., Cordon-Cardo, C., et al. (2001). Inverse associations between plasma lycopene and other carotenoids and prostate cancer. *Cancer Epidemiology Biomarkers Prevention*, 10(7), 749-756.
121. Gann, P.H., Ma, J., Giovannucci, E., Willett, W., Sacks, F.M., Hennekens, C.H., et al. (1999). Lower prostate cancer risk in men with elevated plasma lycopene levels: results of a prospective analysis. *Cancer Research*, 59(6), 1225-1230.
122. Kim, H.S., Bowen, P., Chen, L., Duncan, C., Ghosh, L., Sharifi, R., et al. (2003). Effects of tomato sauce consumption on apoptotic cell death in prostate benign hyperplasia and carcinoma. *Nutrition and Cancer*, 47(1), 40-47.
123. Kucuk, O., Sarkar, F.H., Djuric, Z., Sakr, W., Pollak, M.N., Khachik, F., et al. (2002). Effects of lycopene supplementation in patients with localized prostate cancer. *Experimental Biology and Medicine (Maywood)*, 227(10), 881-885.
124. Bowen, P., Chen, L., Stacewicz-Sapuntzakis, M., Duncan, C., Sharifi, R., Ghosh, L., et al. (2002). Tomato sauce supplementation and prostate cancer: lycopene accumulation and modulation of biomarkers of carcinogenesis. *Experimental Biology and Medicine (Maywood)*, 227(10), 886-893.
125. Chen, L., Stacewicz-Sapuntzakis, M., Duncan, C., Sharifi, R., Ghosh, L., van Breemen, R., et al. (2001). Oxidative DNA damage in prostate cancer patients consuming tomato sauce-based entrees as a whole-food intervention. *Journal of the National Cancer Institute*, 93(24), 1872-1879.
126. Weisburger, J.H. (1998). Evaluation of the evidence on the role of tomato products in disease prevention. *Proceedings of the Society for Experimental Biology and Medicine*, 218(2), 140-143.
127. Boileau, T.W., Liao, Z., Kim, S., Lemeshow, S., Erdman, J.W., Jr, Clinton, S.K. et al. (2003). Prostate carcinogenesis in N-methyl-N-nitrosourea (NMU)-testosterone-treated rats fed tomato powder, lycopene, or energy-restricted diets. *Journal of the National Cancer Institute*, 95(21), 1578-1586.
128. Fraser, M.L., Lee, A.H., & Binns, C.W. (2005). Lycopene and prostate cancer: emerging evidence. *Expert Review of Anticancer Therapy*, 5(5), 847-854.
129. Corcoran, N.M., Najdovska, M., & Costello, A.J. (2004). Inorganic selenium retards progression of experimental hormone refractory prostate cancer. *Journal of Urology*, 171(2 Pt 1), 907-910.
130. Sinha, R., & El-Bayoumy, K. (2004). Apoptosis is a Critical Cellular Event in Cancer Chemoprevention and Chemotherapy by Selenium Compounds. *Current Cancer Drug Targets*, 4(1), 13-28.
131. Clark, L.C., Dalkin, B., Krongrad, A., Combs, G.F. Jr, Turnbull, B.W., Slate, E.H., et al. (1998). Decreased incidence of prostate cancer with selenium supplementation: results of a double-blind cancer prevention trial. *British Journal of Urology*, 81(5), 730-734.
132. Giovannucci, E., Rimm, E.B., Wolk, A., Ascherio, A., Stampfer, M.J., Colditz, G.A., et al. (1998). Calcium and fructose intake in relation to risk of prostate cancer. *Cancer Research*, 58(3), 442-447.
133. Yoshizawa, K., Willett, W.C., Morris, S.J., Stampfer, M.J., Spiegelman, D., Rimm, E.B., et al. (1998). Study of prediagnostic selenium level in toenails and the risk of advanced prostate cancer. *Journal of the National Cancer Institute*, 90(16), 1219-1224.
134. Hartman, T.J., Dorgan, J.F., Woodson, K., Virtamo, J., Tangrea, J.A., Heinonen, O.P., et al. (2001). Effects of long-term alpha-tocopherol supplementation on serum hormones in older men. *Prostate*, 46(1), 33-38.

135. Brooks, J.D., Metter, E.J., Chan, D.W., Sokoll, L.J., Landis, P., Nelson, et al. (2001). Plasma selenium level before diagnosis and the risk of prostate cancer development. *Journal of Urology*, 166(6), 2034-2038.
136. Venkateswaran, V., Fleshner, N.E., & Klotz, L.H. (2004). Synergistic effect of vitamin E and selenium in human prostate cancer cell lines. *Prostate Cancer Prostatic Disease*, 7(1), 54-56.
137. Klein, E.A., Lippman, S.M., Thompson, I.M., Goodman, P.J., Albanes, D., Taylor, P.R., et al. (2003). The selenium and vitamin E cancer prevention trial. *World Journal of Urology*, 21(1), 21-27.
138. Bounous, G., & Molson, J.H. (2003). The antioxidant system. *Anticancer Research*, 23(2B), 1411-1415.
139. Combs, G.F. Jr., Clark, L.C., & Turnbull, B.W. (1997). Reduction of cancer risk with an oral supplement of selenium. *Biomedical Environmental Sciences*, 10(2-3), 227-234.
140. Yoshida, M., Sugihara, S., Suenaga, T., Naito, C., Fukunaga, K., Tsuchita, H., et al. (2002). Digestibility and chemical species of selenium contained in high-selenium yeast. *Journal of Nutritional Science and Vitaminology (Tokyo)*, 48(5), 401-404.
141. Schrauzer, G.N. (2001). Nutritional selenium supplements: product types, quality, and safety. *Journal of the American College of Nutrition*, 20(1), 1-4.
142. Chang, S., Erdman, J.W. Jr, Clinton, S.K., Vadiveloo, M., Strom, S.S., et al. (2005). Relationship between plasma carotenoids and prostate cancer. *Nutrition and Cancer*, 53(2), 127-134.
143. Daviglus, M.L., Dyer, A.R., Persky, V., Chavez, N., Drum, M., Goldberg, J., et al. (1996). Dietary beta-carotene, vitamin C, and risk of prostate cancer: results from the Western Electric Study. *Epidemiology*, 7(5), 472-477.
144. Schuurman, A.G., Goldbohm, R.A., Brants, H.A., & van den Brandt, P.A. (2002). A prospective cohort study on intake of retinol, vitamins C and E, and carotenoids and prostate cancer risk (Netherlands). *Cancer Causes Control*, 13(6), 573-582.
145. Omenn, G.S., Goodman, G.E., Thornquist, M.D., Balmes, J., Cullen, M.R., Glass, A., et al. (1996). Risk factors for lung cancer and for intervention effects in CARET, the Beta-Carotene and Retinol Efficacy Trial. *Journal of the National Cancer Institute*, 88(21), 1550-1559.
146. Hennekens, C.H., Buring, J.E., Manson, J.E., Stampfer, M., Rosner, B., Cook, N.R., et al. (1996). Lack of effect of long-term supplementation with beta carotene on the incidence of malignant neoplasms and cardiovascular disease. *The New England Journal of Medicine*, 334(18), 1145-1149.
147. Heinonen, O.P., Albanes, D., Virtamo, J., Taylor, P.R., Huttunen, J.K., Hartman, A.M., et al. (1998). Prostate cancer and supplementation with alpha-tocopherol and beta-carotene: incidence and mortality in a controlled trial. *Journal of the National Cancer Institute*, 90(6), 440-446.
148. Kristal, A.R., Stanford, J.L., Cohen, J.H., Wicklund, K., & Patterson, R.E. (1999). Vitamin and mineral supplement use is associated with reduced risk of prostate cancer. *Cancer Epidemiology Biomarkers and Prevention*, 8(10), 887-892.
149. Maramba, C., Menon, M., Balaji, K.C., Reddy, P.G., & Laxmanan, S. (1997). Effect of vitamin C on prostate cancer cells in vitro: effect on cell number, viability, and DNA synthesis. *Prostate*, 32(3), 188-195.
150. Helzlsouer, K.J., Huang, H.Y., Alberg, A.J., Hoffman, S., Burke, A., & Norkus, E.P., et al. (2000). Association between alpha-tocopherol, gamma-tocopherol, selenium, and subsequent prostate cancer. *Journal of National Cancer Institute*, 92(24), 2018-2023.
151. Huang, H.Y., Alberg, A.J., Norkus, E.P., Hoffman, S.C., Comstock, G.W., Helzlsouer, K.J., et al. (2003). Prospective study of antioxidant micronutrients in the blood and the risk of developing prostate cancer. *American Journal of Epidemiology*, 157(4), 335-344.
152. Nomura, A.M., Stemmermann, G.N., Lee, J., & Craft, N.E. (1997). Serum micronutrients and prostate cancer in Japanese Americans in Hawaii. *Cancer Epidemiology Biomarkers Prevention*, 6(7), 487-491.
153. Chopra, R.K., & Bhagavan, H.N. (1999). Relative bioavailabilities of natural and synthetic vitamin E formulations containing mixed tocopherols in human subjects. *International Journal of Vitamin Nutrition Research*, 69(2), 92-95.
154. Handelman, G.J., Epstein, W.L., Peerson, J., Spiegelman, D., Machlin, L.J., & Dratz, E.A. (1994). Human adipose alpha-tocopherol and gamma-tocopherol kinetics during and after 1 y of alpha-tocopherol supplementation. *American Journal of Clinical Nutrition*, 59(5), 1025-1032.
155. Handelman, G.J., Machlin, L.J., Fitch, K., Weiter, J.J., & Dratz, E.A. (1985). Oral alpha-tocopherol supplements decrease plasma gamma-tocopherol levels in humans. *Journal of Nutrition*, 115(6), 807-813.
156. Moyad, M.A., Brumfield, S.K., & Pienta, K.J. (1999). Vitamin E, alpha- and gamma-tocopherol, and prostate cancer. *Seminars in Urologic Oncology*, 17(2), 85-90.
157. Zu, K., & Ip, C. (2003). Synergy between selenium and vitamin E in apoptosis induction is associated with activation of distinctive initiator caspases in human prostate cancer cells. *Cancer Research*, 63(20), 6988-6995.
158. Jiang, Q., Christen, S., Shigenaga, M.K., & Ames, B.N. (2001). Gamma-tocopherol, the major form of vitamin E in the US diet, deserves more attention. *American Journal of Clinical Nutrition*, 74(6), 714-722.
159. Galli, F., Stabile, A.M., Betti, M., Conte, C., Pistilli, A., Rende, M., et al. (2004). The effect of alpha- and gamma-tocopherol and their carboxyethyl hydroxychroman metabolites on prostate cancer cell proliferation. *Archives of Biochemistry & Biophysics*, 423(1), 97-102.
160. Sigounas, G., Anagnostou, A., & Steiner, M. (1997). dl-alpha-tocopherol induces apoptosis in erythroleukemia, prostate, and breast cancer cells. *Nutrition and Cancer*, 28(1), 30-35.
161. Malafa, M.P., Fokum, F.D., Andoh, J., Neitzel, L.T., Bandyopadhyay, S., et al. (2006). Vitamin E succinate suppresses prostate tumor growth by inducing apoptosis. *International Journal of Cancer*, 118(10), 2441-2447.
162. Dabrosin, C., Chen, J., Wang, L., & Thompson, L.U. (2002). Flaxseed inhibits metastasis and decreases extracellular vascular endothelial growth factor in human breast cancer xenografts. *Cancer Letters*, 185(1), 31-37.
163. Demark-Wahnefried, W., Price, D.T., Polascik, T.J., Robertson, C.N., Anderson, E.E., Paulson, D.F., et al. (2001). Pilot study of dietary fat restriction and flaxseed supplementation in men with prostate cancer before surgery: exploring the effects on hormonal levels, prostate-specific antigen, and histopathologic features. *Urology*, 58(1), 47-52.
164. Denis, L., Morton, M.S., & Griffiths, K. (1999). Diet and its preventive role in prostatic disease. *European Urology*, 35(5-6), 377-387.
165. Lin, X., Gingrich, J.R., Bao, W., Li, J., Haroon, Z.A., Demark-Wahnefried, W., et al. (2002). Effect of flaxseed supplementation on prostatic carcinoma in transgenic mice. *Urology*, 60(5), 919-924.
166. Moyad, M.A. (2000). The ABCs of nutrition and supplements for prostate cancer. Ann Arbor, MI: JW Edwards Publishing.
167. Hsing, A.W., Chokkalingam, A.P., Gao, Y.T., Madigan, M.P., Deng, J., Gridley, G., et al. (2002). Allium vegetables and risk of prostate cancer: a population-based study. *Journal of the National Cancer Institute*, 94(21), 1648-1651.
168. Pinto, J.T., & Rivlin, R.S. (2001). Antiproliferative effects of allium derivatives from garlic. *The Journal of Nutrition*, 131(3s), 1058S-1060S.
169. Fleischauer, A.T., & Arab, L. (2001). Garlic and cancer: a critical review of the epidemiologic literature. *The Journal of Nutrition*, (3s), 1032S-1040S.

170. Vikse, R., Reistad, R., Steffensen, I.L., Paulsen, J.E., Nyholm, S.H., Alexander, J., et al. (1999). [Heterocyclic amines in cooked meat] [Article in Norwegian] *Tidsskrift for den Norske Lægeforening*, 119(1), 45-49.
171. Shirai, T., Imaida, K., & Ito, N. (2000). Prostate. In: M. Nagao and T. Sugimura (Eds.), *Food Born Carcinogenesis. Current Toxicology Series*, (pp. 270-274). Wiley, Chichester.
172. Shirai, T., Sano, M., Tamano, S., Takahashi, S., Hirose, M., Futakuchi, M., et al. (1997). The prostate: a target for carcinogenicity of 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) derived from cooked foods. *Cancer Research*, 57, 195-198.
173. Cui, L., Takahashi, S., Tada, M., Kato, K., Yamada, Y., Kohri, K., et al. (2000). Immunohistochemical detection of carcinogen-DNA adducts in normal human prostate tissues transplanted into the subcutis of athymic nude mice: Results with 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) and 3,2'-dimethyl-4-aminobiphenyl (DMAB) and relation to cytochrome P450s and N-acetyltransferase activity. *Japanese Journal of Cancer Research*, 91, 52-58.
174. Nowell, S., Ratnasinghe, D.L., Ambrosone, C.B., Williams, S., Teague-Ross, T., Trimble L., et al. (2004). Association of SUL1A1 Phenotype and Genotype with Prostate Cancer Risk in African-Americans and Caucasians. *Cancer Epidemiology Biomarkers Prevention*, 13(2), 270-276.
175. Kooiman, G.G., Martin, F.L., Williams, J.A., Grover, P.L., Phillips, D.H., & Muir, G.H., et al. (2000). The influence of dietary and environmental factors on prostate cancer risk. *Prostate Cancer Prostatic Diseases*, 3(4), 256-258.
176. Hu, J.J., Hall, M.C., Grossman, L., Hedayati, M., McCullough, D.L., Lohman, K., et al. (2004). Deficient nucleotide excision repair capacity enhances human prostate cancer risk. *Cancer Research*, 64(3), 1197-1201.
177. Ferguson, L.R. (2002). Meat consumption, cancer risk and population groups within New Zealand. *Mutation Research*, 506-507, 215-224.
178. Norrish, A.E., Ferguson, L.R., Knize, M.G., Felton, J.S., Sharpe, S.J., & Jackson R.T. (1999). Heterocyclic amine content of cooked meat and risk of prostate cancer. *Journal of the National Cancer Institute*, 91(23), 2038-2044.
179. Eder, E. (1999). Intra-individual variations of DNA adduct levels in humans. *Mutation Research*, 424(1-2), 249-261.
180. Sugimura, T., Wakabayashi, K., Nakagama, H., & Nagao, M. (2004). Heterocyclic amines: Mutagens/carcinogens produced during cooking of meat and fish. *Cancer Sciences*, 95(4), 290-299.
181. Bogen, K.T., & Keating, G.A. (2001). U.S. dietary exposures to heterocyclic amines. *Journal of Exposure Analysis & Environmental Epidemiology*, (3), 155-168.
182. Wilkinson, G.R. (1997). The effects of diet, aging and disease-states on presystemic elimination and oral drug bioavailability in humans. *Advance Drug Delivery Reviews*, 27(2-3), 129-159.
183. Leone, M., Zhai, D., Sareth, S., Kitada, S., Reed, J.C., & Pellecchia, M. (2003). Cancer prevention by tea polyphenols is linked to their direct inhibition of antiapoptotic Bcl-2-family proteins. *Cancer Research*, 63(23), 8118-8121.
184. Takabayashi, F., Tahara, S., Kaneko, T., & Harada, N. (2004). Effect of green tea catechins on oxidative DNA damage of hamster pancreas and liver induced by N-Nitrosobis(2-oxopropyl)amine and/or oxidized soybean oil. *Biofactors*, 21(1-4), 335-337.
185. Gleis, M., & Pool-Zobel, B.L. (2006). The main catechin of green tea, (-)-epigallocatechin-3-gallate (EGCG), reduces bleomycin-induced DNA damage in human leucocytes. *Toxicology In Vitro*, 20(3), 295-300.
186. Stuart, E.C., Scandlyn, M.J., & Rosengren, R.J. (2006). Role of epigallocatechin gallate (EGCG) in the treatment of breast and prostate cancer. *Life Sciences*, 79(25), 2329-2336.
187. Crespy, V., & Williamson, G. (2004). A review of the health effects of green tea catechins in in vivo animal models. *Journal of Nutrition*, 134(12 Suppl), 3431S-3440S.
188. Hussain, T., Gupta, S., Adhami, V.M., & Mukhtar, H. (2005). Green tea constituent epigallocatechin-3-gallate selectively inhibits COX-2 without affecting COX-1 expression in human prostate carcinoma cells. *International Journal of Cancer*, 113(4), 660-669.
189. Patel, S.P., Hotston, M., Kommu, S., & Persad, R.A. (2005). Green tea constituent epigallocatechin-3-gallate selectively inhibits COX-2 without affecting COX-1 expression in human prostate carcinoma cells. *International Journal of Cancer*, 113(4), 660-669.
190. Saleem, M., Adhami, V.M., Siddiqui, I.A., & Mukhtar, H. (2003). Tea beverage in chemoprevention of prostate cancer: a mini-review. *Nutrition Cancer*, 47(1), 13-23.
191. Gupta, S., Hussain, T., & Mukhtar, H. (2003). Molecular pathway for (-)-epigallocatechin-3-gallate-induced cell cycle arrest and apoptosis of human prostate carcinoma cells. *Archives of Biochemistry and Biophysics*, 410(1), 177-185.
192. Jian, L., Xie, L.P., Lee, A.H., & Binns, C.W. (2004). Protective effect of green tea against prostate cancer: a case-control study in southeast China. *International Journal of Cancer*, 108(1), 130-135.
193. Lee, A.H., Fraser, M.L., Meng, X., & Binns, C.W. (2006). Protective effects of green tea against prostate cancer. *Expert Reviews of Anticancer Therapies*, 6(4), 507-513.
194. Yu, H.N., Yin, J.J., & Shen, S.R. (2004). Growth inhibition of prostate cancer cells by epigallocatechin gallate in the presence of Cu²⁺. *Journal of Agricultural Food Chemistry*, 52(3), 462-466.
195. Brusselmans, K., De Schrijver, E., Heyns, W., Verhoeven, G., & Swinnen, J.V. (2003). Epigallocatechin-3-gallate is a potent natural inhibitor of fatty acid synthase in intact cells and selectively induces apoptosis in prostate cancer cells. *International Journal of Cancer*, 106(6), 856-862.
196. Chung, L.Y., Cheung, T.C., Kong, S.K., Fung, K.P., Choy, Y.M., Chan, Z.Y., et al. (2001). Induction of apoptosis by green tea catechins in human prostate cancer DU145 cells. *Life Sciences*, 68(10), 1207-1214.
197. Hastak, K., Gupta, S., Ahmad, N., Agarwal, M.K., Agarwal, M.L., Mukhtar, H., et al. (2003). Role of p53 and NF-kappaB in epigallocatechin-3-gallate-induced apoptosis of LNCaP cells. *Oncogene*, 22(31), 4851-4859.
198. Gupta, S., Srivastava, M., Ahmad, N., Sakamoto, K., Bostwick, D.G., Mukhtar, H., et al. (2001). Lipoxigenase-5 is overexpressed in prostate adenocarcinoma. *Cancer*, 91(4), 737-743.
199. Kikuchi, N., Ohmori, K., Shimazu, T., Nakaya, N., Kuriyama, S., et al. (2006). No association between green tea and prostate cancer risk in Japanese men: the Ohsaki Cohort Study. *British Journal of Cancer*, 95(3), 371-373.
200. Zhou, J.R., Yu, L., Zhong, Y., & Blackburn, G.L. (2003). Soy phytochemicals and tea bioactive components synergistically inhibit androgen-sensitive human prostate tumors in mice. *Journal of Nutrition*, 133(2), 516-521.
201. Jatoi, A., Ellison, N., Burch, P.A., Sloan, J.A., Dakhlil, S.R., Novotny, P., et al. (2003). A phase II trial of green tea in the treatment of patients with androgen independent metastatic carcinoma. *Cancer*, 97(6), 1442-1446.
202. Malik, A., Afaq, F., Sarfaraz, S., Adhami, V.M., Syed, D.N., et al. (2005). Pomegranate fruit juice for chemoprevention and chemotherapy of prostate cancer. *Proceedings of the National Academy of Sciences USA*, 102(41), 14813-14818.

203. Toi, M., Bando, H., Ramachandran, C., Melnick, S.J., Imai, A., Fife, R.S., et al. (2003). Preliminary studies on the anti-angiogenic potential of pomegranate fractions in vitro and in vivo. *Angiogenesis*, 6(2), 121-128.
204. Pantuck, A.J., Leppert, J.T., Zomorodian, N., Aronson, W., Hong, J., et al. (2006). Phase II study of pomegranate juice for men with rising prostate-specific antigen following surgery or radiation for prostate cancer. *Clinical Cancer Research*, 12(13), 4018-4026.
205. Jenkins, D.J., Kendall, C.W., D'Costa, M.A., Jackson, C.J., Vidgen, E., Singer, W., et al. (2003). Soy consumption and phytoestrogens: effect on serum prostate specific antigen when blood lipids and oxidized low-density lipoprotein are reduced in hyperlipidemic men. *Journal of Urology*, 169(2), 507-511.
206. McCarty, M.F. (1999). Vegan proteins may reduce risk of cancer, obesity, and cardiovascular disease by promoting increased glucagon activity. *Medical Hypotheses*, 53(6), 459-485.
207. Arliss, R.M., & Biermann, C.A. (2002). Do soy isoflavones lower cholesterol, inhibit atherosclerosis, and play a role in cancer prevention? *Holistic Nursing Practice*, 16(5), 40-48.
208. Setchell, K.D., & Lydeking-Olsen, E. (2003). Dietary phytoestrogens and their effect on bone: evidence from in vitro and in vivo, human observational, and dietary intervention studies. *American Journal of Clinical Nutrition*, 78(3 Suppl), 593S-609S.
209. Ho, S.C., Woo, J., Lam, S., Chen, Y., Sham, A., Lau, J., et al. (2003). Soy protein consumption and bone mass in early postmenopausal Chinese women. *Osteoporosis International*, 14(10), 835-842.
210. Messina, M.J., Persky, V., Setchell, K.D., & Barnes, S. (1994). Soy intake and cancer risk: a review of the in vitro and in vivo data. *Nutrition Cancer*, 21, 113-131.
211. Zhou, J.R., Yu, L., Zhong, Y., Nassr, R.L., Franke, A.A., Gaston S.M., et al. (2002). Inhibition of orthotopic growth and metastasis of androgen-sensitive human prostate tumors in mice by bioactive soybean components. *Prostate*, Oct 1, 53(2), 143-153.
212. Messina, M.J. (2003). Emerging evidence on the role of soy in reducing prostate cancer risk. *Nutrition Reviews*, 61(4), 117-131.
213. Pollard, M., & Wolter, W. (2000). Prevention of spontaneous prostate-related cancer in Lobund-Wistar rats by a soy protein isolate/isoflavone diet. *Prostate*, 45(2), 101-105.
214. Jacobsen, B.K., Knutsen, S.F., & Fraser, G.E. (1998). Does high soy milk intake reduce prostate cancer incidence? The Adventist Health Study (United States). *Cancer Causes & Control*, 9, 553-557.
215. Aronson, W.J., Tymchuk, C.N., Elashoff, R.M., McBride, W.H., McLean, C., Wang, H., et al. (1999). Decreased growth of human prostate LNCaP tumors in SCID mice fed a low-fat, soy protein diet with isoflavones. *Nutrition and Cancer*, 35(2), 130-136.
216. Yan, L., & Spitznagel, E.L. (2005). Meta-analysis of soy food and risk of prostate cancer in men. *International Journal of Cancer*, 117(4), 667-669.
217. Yi, M.A., Son, H.M., Lee, J.S., Kwon, C.S., Lim, J.K., Yeo, Y.K., et al. (2002). Regulation of male sex hormone levels by soy isoflavones in rats. *Nutrition & Cancer*, 42(2), 206-210.
218. Gardner-Thorpe, D., O'Hagen, C., Young, I., & Lewis, S.J. (2003). Dietary supplements of soya flour lower serum testosterone concentrations and improve markers of oxidative stress in men. *European Journal of Clinical Nutrition*, 57(1), 100-106.
219. Tsutsumi, M., Suzuki, K., Shiga, Y., Ishikawa, S., & Ishikawa, Y. (2002). [A low-fat and high soybean protein diet for patients with elevated serum PSA level: alteration of QOL and serum PSA level after the dietary intervention] [Article in Japanese] *Hinyokika Kiyo*, 48(4), 207-211.
220. Moyad, M.A., Sakr, W.A., Hirano, D., & Miller, G.J. (2001). Complementary medicine for prostate cancer: effects of soy and fat consumption. *Reviews of Urology*, 3 Suppl 2, S20-S30.
221. Messina, M., Kucuk, O., & Lampe, J.W. (2006). An overview of the health effects of isoflavones with an emphasis on prostate cancer risk and prostate-specific antigen levels. *Journal of AOAC International*, 89(4), 1121-1134.
222. Getzenberg, R.H., Light, B.W., Lapco, B.W., Konety B.R., Nangia A.K., Acierno J.S., et al. (1997). Vitamin D inhibition of prostate adenocarcinoma growth and metastasis in the Dunning rat prostate model system. *Urology*, 50, 999-1006.
223. Schwartz, G.G., Oeler, T.A., Uskokovic, M.R., & Bahnsen, R.R. (1994). Human prostate cancer cells: inhibition of proliferation by vitamin D analogs. *Anticancer Research*, 14, 1077-1081.
224. Lokeshwar, B.L., Schwartz, G.G., Selzer, M.G., Burnstein, K.L., Zhuang, S.H., & Block, N.L., et al. (1999). Inhibition of prostate cancer metastasis in vivo: a comparison of 1,23-dihydroxyvitamin D (calcitriol) and EB1089. *Cancer Epidemiology Biomarkers Prevention*, 8(3), 241-248.
225. Blutt, S.E., Polek, T.C., Stewart, L.V., Kattan, M.W., & Weigel, N.L. (2000). A calcitriol analogue, EB1089, inhibits the growth of LNCaP tumors in nude mice. *Cancer Research*, 60(4), 779-782.
226. Vegesna, V., O'Kelly, J., Said, J., Uskokovic, M., Binderup, L., Koeffle, H.P., et al. (2003). Ability of potent vitamin D3 analogs to inhibit growth of prostate cancer cells in vivo. *Anticancer Research*, 23(1A), 283-289.
227. Ahonen, M.H., Tenkanen, L., Teppo, L., Hakama, M., & Tuohimaa, P. (2000). Prostate cancer risk and prediagnostic serum 25-hydroxyvitamin D levels (Finland). *Cancer Causes Control*, 11(9), 847-852.
228. Tuohimaa, P., Tenkanen, L., Ahonen, M., Lumme, S., Jellum, E., Hallmans, G., et al. (2004). Both high and low levels of blood vitamin D are associated with a higher prostate cancer risk: a longitudinal, nested case-control study in the Nordic countries. *International Journal of Cancer*, 108(1), 104-108.
229. Peehl, D.M., Krishnan, A.V., & Feldman, D. (2003). Pathways mediating the growth-inhibitory actions of vitamin D in prostate cancer. *Journal of Nutrition*, 133(7 Suppl), 2461S-2469S.
230. Studzinski, G.P., & Moore, D.C. (1995). Sunlight--can it prevent as well as cause cancer? *Cancer Research*, 55(18), 4014-4022.
231. Gross, C., Stamey, T., Hancock, S., & Feldman, D. (1998). Treatment of early recurrent prostate cancer with 1,25-dihydroxyvitamin D3 (calcitriol) *Journal of Urology*, 159(6), 2035-2039.
232. Chen, T.C., Wang, L., Whitlatch, L.W., Flanagan, J.N., & Holick, M.F. (2003). Prostatic 25-hydroxyvitamin D-1alpha-hydroxylase and its implication in prostate cancer. *Journal of Cell Biochemistry*, 88(2), 315-322.
233. Hanchette, C.L., & Schwartz, G.G. (1992). Geographic patterns of prostate cancer mortality. Evidence for a protective effect of ultraviolet radiation. *Cancer*, 70(12), 2861-2869.
234. Corder, E.H., Guess, H.A., Hulka, B.S., Friedman, G.D., Sadler, M., Vollmer, R.T., et al. (1993). Vitamin D and prostate cancer: a prediagnostic study with stored sera. *Cancer Epidemiology, Biomarkers and Prevention*, 2(5), 467-472.
235. Giovannucci, E., Liu, Y., Rimm, E.B., Hollis, B.W., Fuchs, C.S., et al. (2006). Prospective study of predictors of vitamin D status and cancer incidence and mortality in men. *Journal of the National Cancer Institute*, 98(7), 451-459.
236. Beer, T.M. (2003). Development of weekly high-dose calcitriol based therapy for prostate cancer. *Urology Oncology*, 21(5), 399-405.
237. Grant, W.B. (1999). An ecologic study of dietary links to prostate cancer. *Alternative Medicine Reviews*, (3), 162-169.

238. Giovannucci, E. (2005). The epidemiology of vitamin D and cancer incidence and mortality: a review (United States). *Cancer Causes Control*, 16(2), 83-95.
239. Gao, X., LaValley, M.P., & Tucker, K.L. (2005). Prospective studies of dairy product and calcium intakes and prostate cancer risk: a meta-analysis. *Journal of the National Cancer Institute*, 97(23), 1768-1777.
240. Kesse, E., Bertrais, S., Astorg, P., Jauoen, A., & Arnault, N. (2006). Dairy products, calcium and phosphorus intake, and the risk of prostate cancer: results of the French prospective SU.VI.MAX (Supplementation en Vitamines et Mineraux Antioxydants) study. *British Journal of Nutrition*, 95(3), 539-545.
241. Tseng, M., Breslow, R.A., Graubard, B.I., & Ziegler, R.G. (2005). Dairy, calcium, and vitamin D intakes and prostate cancer risk in the National Health and Nutrition Examination Epidemiologic Follow-up Study cohort. *American Journal of Clinical Nutrition*, 81(5), 1147-1154.
242. Giovannucci, E., Liu, Y., Stampfer, M.J., & Willett, W.C. (2006). A prospective study of calcium intake and incident and fatal prostate cancer. *Cancer Epidemiology and Biomarkers Prevention*, 15(2), 203-210.
243. Gunnell, D., Oliver, S.E., Peters, T.J., Donovan, J.L., Persad, R., Maynard, M., et al. (2003). Are diet-prostate cancer associations mediated by the IGF axis? A cross-sectional analysis of diet, IGF-I and IGFBP-3 in healthy middle-aged men. *British Journal of Cancer*, 88(11), 1682-1686.
244. Holick, M.F. (1995). Defects in the synthesis and metabolism of vitamin D. *Experimental Clinical Endocrinology Diabetes*, 103(4), 219-227.
245. Thomas, M.K., Lloyd-Jones, D.M., Thadhani, R.I., Shaw, A.C., Deraska, D.J., Kitch, B.T., et al. (1998). Hypovitaminosis D in medical inpatients. *New England Journal of Medicine*, 338, 777-778.
246. Rasmussen, L.B., Hansen, G.L., Hansen, E., Koch, B., Mosekilde, L., Molgaard, C., et al. (2000). Vitamin D: should the supply in the Danish population be increased? *International Journal of Food Science Nutrition*, 51(3), 209-215.
247. Webb, A.R., Pilbeam, C., Hanafin, N., & Holick, M.F. (1990). An evaluation of the relative contributions of exposure to sunlight and of diet to the circulating concentrations of 25-hydroxyvitamin D in an elderly nursing home population in Boston. *American Journal of Clinical Nutrition*, 51, 1075-1081.
248. Silverberg, S.J., Shane, E., dela Cruz, L., Segre, C.V., Clemens, T.L., Bilezikian, J.P., et al. (1989). Vitamin D hydroxylation abnormalities in parathyroid hormone secretion and 1,25-dihydroxyvitamin D-3 formation in women with osteoporosis. *New England Journal of Medicine*, 320, 277-281.
249. Ebeling, P.R., Sandgren, M.E., DiMagno, E.P., Lane, A.W., DeLuca, H.F., Riggs B.L., et al. (1992). Evidence of an age-related decrease in intestinal responsiveness to vitamin D: relationship between serum 1,25-dihydroxyvitamin D-3 and intestinal vitamin D receptor concentrations in normal women. *Journal of Clinical Endocrinology Metabolism*, 75, 176-182.
250. Bischoff-Ferrari, H.A., Giovannucci, E., Willett, W.C., Dietrich, T., & Dawson-Hughes, B. (2006). Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. *American Journal of Clinical Nutrition*, 84(1), 18-28.
251. Singh, R.P., & Agarwal, R. (2004). Prostate cancer prevention by silibinin. *Current Cancer Drug Targets*, 4(1), 1-11.
252. Singh, R.P., Dhanalakshmi, S., Tyagi, A.K., Chan, D.C., Agarwal, C., & Agarwal, R. (2002). Dietary feeding of silibinin inhibits advance human prostate carcinoma growth in athymic nude mice and increases plasma insulin-like growth factor-binding protein-3 levels. *Cancer Research*, 62(11), 3063-3069.
253. Tyagi, A., Bhatia, N., Condon, M.S., Bosland, M.C., Agarwal, C., & Agarwal, R. (2002). Antiproliferative and apoptotic effects of silibinin in rat prostate cancer cells. *Prostate*, 53(3), 211-217.
254. Tyagi, A.K., Singh, R.P., Agarwal, C., Chan, D.C., & Agarwal, R. (2002). Silibinin strongly synergizes human prostate carcinoma DU145 cells to doxorubicin-induced growth inhibition, G2-M arrest, and apoptosis. *Clinical Cancer Research*, 8(11), 3512-3519.
255. Zi, X., Zhang, J., Agarwal, R., & Pollak, M. (2000). Silibinin up-regulates insulin-like growth factor-binding protein 3 expression and inhibits proliferation of androgen-independent prostate cancer cells. *Cancer Research*, 60(20), 5617-5620.
256. Yip, I., Cudiamat, M., & Chim, D. (2003). PC-SPES for treatment of prostate cancer: herbal medicine. *Current Urology Reports*, 4(3), 253-257.
257. Cordell, G.A. (2002). PC-SPES: a brief overview. *Integrative Cancer Therapies*, 1(3), 271-286.
258. Katz, A.E. (2002). Flavonoid and botanical approaches to prostate health. *Journal of Alternative and Complementary Medicine*, 8(6), 813-821.
259. Goldmann, W.H., Sharma, A.L., Currier, S.J., Johnston, P.D., Rana, A., & Sharma, C.P. (2001). Saw palmetto berry extract inhibits cell growth and Cox-2 expression in prostatic cancer cells. *Cell Biology International*, 25(11), 1117-1124.
260. Dorai, T., Cao, Y.C., Dorai, B., Buttyan, R., & Katz, A.E. (2001). Therapeutic potential of curcumin in human prostate cancer. III. Curcumin inhibits proliferation, induces apoptosis, and inhibits angiogenesis of LNCaP prostate cancer cells in vivo. *Prostate*, 47(4), 293-303.
261. Dorai, T., Gehani, N., & Katz, A. (2000). Therapeutic potential of curcumin in human prostate cancer. II. Curcumin inhibits tyrosine kinase activity of epidermal growth factor receptor and depletes the protein. *Molecular Urology*, 4(1), 1-6.
262. Deeb, D., Xu, Y.X., Jiang, H., Gao, X., Janakiraman, N., Chapman, R.A., et al. (2003). Curcumin (diferuloyl-methane) enhances tumor necrosis factor-related apoptosis-inducing ligand-induced apoptosis in LNCaP prostate cancer cells. *Molecular Cancer Therapeutics*, 2(1), 95-103.
263. Chendil, D., Ranga, R.S., Meigooni, D., Sathishkumar, S., & Ahmed, M.M. (2004). Curcumin confers radiosensitizing effect in prostate cancer cell line PC-3. *Oncogene*, 23(8), 1599-1607.
264. Guo, H., Yu, J.H., Chen, K., Ye, Z.Q., & Liu, G.C. (2006). [Curcumin-induced apoptosis in androgen-dependent prostate cancer cell line LNCaP in vitro] [Article in Chinese] *Zhonghua Nan Ke Xue*, 12(2), 141-144.
265. Huang, M.T., Lou, Y.R., Xie, J.G., Ma, W., Lu, Y.P., Yen, P., et al. (1998). Effect of dietary curcumin and dibenzoylmethane on formation of 7,12-dimethylbenz[a]anthracene-induced mammary tumors and lymphomas/leukemias in Sencar mice. *Carcinogenesis*, 19(9), 1697-1700.
266. Wallace, J.M. (2002). Nutritional and botanical modulation of the inflammatory cascade--eicosanoids, cyclooxygenases, and lipoxygenases--as an adjunct in cancer therapy. *Integrative Cancer Therapies*, 1(1), 7-37.
267. Bemis, D.L., Capodice, J.L., Anastasiadis, A.G., Katz, A.E., & Buttyan, R. (2005). Zylflamend, a unique herbal preparation with nonselective COX inhibitory activity, induces apoptosis of prostate cancer cells that lack COX-2 expression. *Nutrition and Cancer*, 52(2), 202-212.
268. Thompson, C.A., Shanafelt, T.D., & Loprinzi, C.L. (2003). Andropause: symptom management for prostate cancer patients treated with hormonal ablation. *Oncologist*, 8(5), 474-487.

269. Van de Weijer, P.H., & Barentsen, R. (2002). Isoflavones from red clover (Promensil) significantly reduce menopausal hot flush symptoms compared with placebo. *Maturitas*, 42(3), 187-193.
270. Hernandez Munoz, G., & Pluchino, S. (2003). Cimicifuga racemosa for the treatment of hot flushes in women surviving breast cancer. *Maturitas*, 44 (Suppl 1), S59-S65.
271. Messina, M., & Hughes, C. (2003). Efficacy of soyfoods and soybean isoflavone supplements for alleviating menopausal symptoms is positively related to initial hot flush frequency. *Journal of Medicinal Food*, 6(1), 1-11.
272. Smith, M.R.(2004). Changes in fat and lean body mass during androgen-deprivation therapy for prostate cancer. *Urology*, 63(4), 742-745.
273. Smith, M.R. (2002). Osteoporosis and other adverse body composition changes during androgen deprivation therapy for prostate cancer. *Cancer and Metastasis Reviews*, 21(2), 159-166.
274. Berruti, A., Dogliotti, L., Terrone, C., Cerutti, S., Isaia, G., Tarabuzzi, R., et al. (2002). Changes in bone mineral density, lean body mass and fat content as measured by dual energy x-ray absorptiometry in patients with prostate cancer without apparent bone metastases given androgen deprivation therapy. *Journal of Urology*, 167(6), 2361-2367.
275. Basaria, S., Lieb, J. 2nd, Tang, A.M., DeWeese, T., Carducci, M., Eisenberger, M., et al. (2002). Long-term effects of androgen deprivation therapy in prostate cancer patients. *Clinical Endocrinology*, 56(6), 779-786.
276. Nowicki, M., Bryc, W., & Kokot, F. (2001). Hormonal regulation of appetite and body mass in patients with advanced prostate cancer treated with combined androgen blockade. *Journal of Endocrinological Investigation*, 24(1), 31-36.
277. Kiratli, B.J., Srinivas, S., Perkash, I., & Terris, M.K. (2001). Progressive decrease in bone density over 10 years of androgen deprivation therapy in patients with prostate cancer. *Urology*, 57, 127-132.
278. Daniell, H.W., Dunn, S.R., Ferguson, D.W. Lomas, G., Niazi, Z., Stratte, P.T., et al. (2000). Progressive osteoporosis during androgen deprivation therapy for prostate cancer. *Journal of Urology*, 163, 181-186.
279. Mittan, D., Lee, S., Miller, E. Perez, R.C., Basler, J.W., Bruder, J.M., et al. (2002). Bone loss following hypogonadism in men with prostate cancer treated with GnRH analogs. *The Journal of Clinical Endocrinology and Metabolism*, 87, 3656-3661.
280. Maillefert, J.F., Sibilia, J., Michel, F. Saussine, C., Javier, R.M., Tavernier, C., et al. (1999). Bone mineral density in men treated with synthetic gonadotropin-releasing hormone agonists for prostatic carcinoma. *Journal of Urology*, 161, 1219-1222.
281. Stoch, S.A., Parker, R.A., Chen, L., Buble, G., Ko, Y.J., Vincelette, A., et al. (2001). Bone loss in men with prostate cancer treated with gonadotropin-releasing hormone agonists. *Journal of Clinical Endocrinology and Metabolism*, 86, 2787-2791.
282. Wei, J.T., Gross, M., Jaffe, C.A. Gravlín, K., Lahaie, M., Faerber, G.J., et al. (1999). Androgen deprivation therapy for prostate cancer results in significant loss of bone density. *Urology*, 54, 607-611.