Nutritional intervention for cancer minimization

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Diet has been linked to the pathogenesis of one in three cancers. Cancer remains a leading cause of death in contemporary society. Alteration to dietary habits may be helpful in reducing an individual's risk of neoplasia. This paper examines how nutritional advice may be used as a cancer preventive measure in chiropractic clinics. (JCCA 1987; 31(4): 185–196)

KEY WORDS: cancer, prevention, diet, chiropractic.

L'alimentation a été liée à la pathogénèse d'un cancer sur trois. Le cancer demeure une des causes premières de décès dans notre société contemporaine. Un changement des habitudes alimentaires peut aider à réduire le risque de néoplasie chez un individu. Cet article étudie la façon d'utiliser les conseils nutritionnels comme mesure préventive du cancer dans les cliniques chiropratiques. (JCCA 1987; 31(4): 185–196)

MOTS CLÉS: cancer, prévention, alimentation, chiropractique.

Introduction

Cancer is responsible for one quarter of all deaths amongst adult Australian males; it kills one in five women over the age of 60 and accounts for nine out of every 20 deaths in women aged 20–59.[1] The major cancers causing death in Australian adults in Victoria are those of the lung, breast, colon and prostate.[2] While smoking remains the single greatest cause implicated in the aetiology of lung cancer, all the cancers listed are believed to be influenced by dietary habits.

The postulated links between diet and cancer are not confined to the ingestion of carcinogens. Both macro- and micronutrients are believed to influence carcinogenesis. Nutrients are hypothesised to influence carcinogenesis directly and indirectly via modulation of both intermediary metabolism and the immune system. Nutrient selection, it is postulated, can either increase or reduce the individual's risk of cancer. It has been estimated that 35% (range 10–70%) of cancers diagnosed in the United States may be attributable to diet.[3]

Epidemiological evidence supports a role for diet in the pathogenesis of cancer.[4] Dietary factors may in part explain the acquisition by migrant populations of the local population's cancer incidence rates. Diet may also help to explain the discrepancies in cancer incidence rates when comparing socioeconomically developed with poor countries – the correlation between high fat diets and colonic and/or breast carcinoma is >0.8. A clear causal relationship between particular dietary constituents and cancer is however difficult to determine. While the potential contribution of retrospective dietary studies is perceived as great, the validity of such studies is somewhat questionable.[5] Prospective studies are therefore desirable. In view of the prolonged delay between exposure to carcinogens and the clinical manifestation of cancer, as well as the difficulties inherent in accurate dietary intake studies, it is unlikely that definitive evidence causally linking particular nutrients to specific neoplasms will be readily available in the foreseeable future. It is therefore desirable that currently available information be critically used in the clinical situation.

Cancer is a disease with a significant mortality and morbidity. Primary contact practitioners have a role to play in both attempting to prevent and in ensuring early diagnosis of this disease. Intervention by chiropractors may be aimed at identifying individual's at risk of developing carcinoma and at early diagnosis, i.e. case finding, in those who already have cancer. Intervention in the former may be totally carried out in the chiropractic clinic; therapy for the latter requires referral. This paper focuses on the use of nutrition as a cancer prevention measure in chiropractic clinics.

The natural history of neoplasia

In order that attempts to minimize the risk of carcinogenesis by nutritional means be logical, it is important that the practitioner have a clear understanding of the postulated mechanisms underlying carcinogenesis.

Neoplasia is a multifactorial, multistage process which progresses through a sequence of changes in response to environmental determinants interacting with the individual's genetic composition.[6,7] Environmental stimuli which act as initiators (mutagens) irreversibly alter the genome of the cell. Uncontrolled cellular proliferation may result should this mutated cell be exposed to one or more promoting agents. Before healthy cells become pleomorphic and demonstrate the features characteristic of anaplastic tissue, they require exposure to both initiating and promoting agents. While initiating and promoting chemicals may, in certain instances, be the same compound, the latter requires prolonged exposure before cellular transformation is established. Initiation, because it involves genetic mutation, is irreversible; promotion is not. Chemical, including dietary, carcinogenesis is theoretically preventable should appropriate intervention be targeted at promoting agents.

Dietary intervention in the prevention of carcinogenesis may therefore be aimed at:

1 the avoidance of carcinogens in ingested foods. Initiators include alcohol, nitrosamines, amino acid pyrolysates, polycyclic hydrocarbons, fat and aflatoxins.
2 The minimization of production of carcinogens during digestion and metabolism. Promoting agents may be neutralized by vitamin E, C, A or beta carotene, selenium, fiber and limitation on energy intake are also believed to be helpful at this level.
3 Optimization of immune function. Nutritional deficiency or

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excess can impair normal function of the immune system.[8] The immune system has been postulated to perform a surveillance function and eliminate abnormal cells.

Ingestion and absorption of carcinogens

Dietary carcinogens abound in nature. Plants synthesise toxic materials as a defence against bacterial, fungal and insect attack. Mutagens found in plants include anthraquinone derivatives found in rhubarb; pyrrolizidine alkaloids encountered in herbs, herbal teas and some honeys, and linear furocoumarins widespread in the parsnip, fig, celery and parsley family.[9] Enhanced production of these "plant protective" human toxins frequently occurs when the plant or its fruit is damaged. Food contaminated by moulds, eg. peanuts, bread, cheese, fruit or corn, may also be carcinogenic. Examples of carcinogenic fungal toxins include aflatoxin, luteoskyrin and sterigmatocystin.[10]

In addition to de novo carcinogens, normal food constituents may acquire carcinogenic activity during processing or cooking.[11] Carcinogens are produced when amino acids and sugars are involved in caramilization or browning. Burned foods, including coffee, contain mutagenic compounds. Charred foods are best avoided; a two packet a day smoker inhales about 0.5 grams of burnt material (using 20mg tar cigarettes), more than one gram of browned and burnt material may be ingested each day. Both smokers and persons who have eaten a meal of fried pork or bacon have mutagens detectable in their urine.

Innoxious food constituents may acquire mutagenic or promotion properties as a result of digestion or bacterial metabolism within the gastro-intestinal tract. Bacterial catabolism of amino acids, eg. tyrosine or tryptophan, within the intestine may produce carcinogenic products.[12] Ingested nitrites and nitrates may be converted to nitrosamines and nitroso compounds within the gastro-intestinal tract. Beets, celery, lettuce, spinach, radishes and rhubarb each contain about 200mg of nitrate per 100 gram portion. Conversion of foodsubstances to nitosamines is impaired by the acidity of the stomach; persons with achlorrhydria are therefore at increased risk. It has also been found that vitamin C, and to a lesser extent, vitamin E can largely block nitrosation of substrates.

Colonic protection against carcinogens in the diet may, to some extent, be achieved by dietary fibre. The recommended daily intake of dietary fibre is 35 grams.[13] Addition of fibre at these levels to high protein diet may result in negative calcium balance.[14] The duration of such negative balance was not studied. Fibre may reduce exposure of the colonic mucosa to carcinogens by altering the bacterial flora, binding bile salts and decreasing the transit time of intestinal contents.[15]

Carcinogenic intermediary metabolites

Intermediary metabolism may also prove a source of carcinogenic products. Alcohol metabolism results in production of acetaldehyde, a mutagen and co-carcinogen. In an aerobic cellular environment, energy production and peroxidation of unsaturated fatty acids may involve formation of free radicals. Unless adequately controlled free radicals can cause tissue damage, including anaplasia.[16]

A free radical is a molecule containing an odd number of electrons; such molecules are chemically unstable and highly reactive.[17] Compared to enzymatic metabolism free-radical processes tend to be irreversible, non-homeostatic, non-cyclic and energetically wasteful.[18] Free radicals are generated during aerobic metabolism including oxidative phosphorylation with energy (ATP) production, intermediary metabolism involving oxidases or dehydrogenases and during the oxygen burst of phagocytosis. It is thus possible, to a certain degree, to enhance free radical generation by increasing ones intake of calories, especially increasing ones intake of fat (an energy dense nutrient) and by experiencing tissue injury.

Once generated free radicals are usually rapidly neutralized. Free radicals which are not quenched may react with various molecules, eg. with DNA thereby modifying genetic information, or with lipids thereby altering cell membrane integrity. It has been hypothesised that free radicals may play a role in the initiation and/or promotion of carcinogenesis.[19] This hypothesis finds some support in the theory of carcinogenesis which proposes that carcinoma only results as a result of failure of normal immunological surveillance; it is also congruent with the finding that certain cancers have a positive correlation with increased fat/energy intake. In both instances alternative explanations are possible.

The quenching of free radicals is at least as an important variable in the pathogenesis of disease as their generation. Two systems of defence have evolved in oxygen utilizing organisms. These are 1) the enzyme system exemplified by superoxide dismutase, catalase and glutathione peroxidase which detoxifies oxygen derived free radicals and 2) the scavenger system, which neutralizes free radicals by the donation of electrons thereby stabilizing oxygen.[20] Free radical scavengers include vitamin C, E and A (retinoids & Beta carotene). One prospective study of dietary intake and of serum levels of vitamins A and E failed to demonstrate a link between these antioxidants and the risk of developing cancers[21]; another study suggested that low serum vitamin E levels do correlate with an increased risk of breast cancer.[22] Further investigation is required. Vitamin C as an antitumour agent enjoys similar controversy.[23,24]

In vitro testing of plasma has demonstrated that it has anti-oxidant activity. This anti-oxidant activity has been linked to caeruloplasmin (a copper containing protein) and transferrin (an iron containing protein). Both of these serum proteins contain minerals with oxidized and reduced forms. Trace elements, recognised as having anti-oxidant activity, have also been identified in various superoxide dismutases (zinc, magnesium, also copper) and glutathione peroxidase (selenium). The protective effects of high dietary selenium may however be unrelated to its physiological role in this enzyme. Selenium has been found to protect against chemical induction of mammary cancer in mice,[25] to reduce the mutagenicity of benzanthracene

derivatives in the Ames test[26] and to impair tumour transplantability.[27]

It has also been suggested that certain foods may enhance free radical production and carcinogenesis. Polyunsaturated fatty acids have been shown to be more effective in inducing mammary, intestinal and pancreatic tumours in experimental animals than saturated fats, a less chemically reactive species.[29] These finding have not been confirmed in man; on the contrary, eskimo studies suggest that dietary fish oils do not promote cancer and may be inhibitory. Speculation about a difference in the carcinogenic potential of omega-3 and omega-6 fatty acids deserves investigation.

Another corollary of the free radical theory of carcinogenesis as this relates to antioxidants is that certain antioxidant food additives may protect against chemically induced cancer. BHT and BHA are antioxidants and therefore may stabilize potential co-carcinogen or carcinogen precusors such as polyunsaturated fatty acids and act as genotoxic free radical scavengers. The major protective effect against chemically induced neoplasia in animals of high concentrations of these additives in foods does however appear to be achieved by enhancing the catabolism and excretion of carcinogens.[29]

Diet and immunity

Malnutrition affects the host's immune system; both cellular and humeral immunity are impaired.[30] While cell-mediated immunity is particularly compromised; the efficiency of antibody production, phagocytosis and the complement system are all reduced in states of inadequate nutrition.[31] Cell mediated immunity is markedly depressed when dietary deprivation of protein-calories, folic acid, iron, zinc or pyridoxine occurs.[32,33] Competent cell mediated immunity may be a prerequisite to adequate cancer surveillance.

Nutrient supplementation in persons on adequate diets may also influence immunity. Polyunsaturated fatty acids are postulated to have an immuno-regulatory function; vitamin E supplementation is believed to initially stimulate and at higher concentrations to impair immune functions, vitamin A supplements are thought to have an adjuvant like effect on immunity while megadoses of vitamin C have been demonstrated to markedly alter phagocytosis.[34]

While nutritional inadequacy indisputably has implications for the immune system, it is far from clear whether nutritional supplementation, beyond that of recommended daily allowances, has any beneficial effect on immunity with regard to subsequent cancer susceptibility.

Dietary intervention to reduce the risk of cancer

Diet and cancer are linked. Certain dietary constituents may increase the risk of carcinogenesis; careful dietary selection may reduce an individual's personal risk. The interim dietary guidelines of the Committee on Diet, Nutrition and Cancer of the National Academy of Sciences suggest a reduction in fat consumption (no more than 30% of total calories) with moderation in alcohol intake and minimization of consumption of foods preserved by self-curing or smoking. They recommend an increase in whole grains, fruit and vegetables and an ongoing effort to identify and exclude carcinogens and mutagens from foodsubstances.[35]

Dietary factors which appear to increase the risks of carcinoma and therefore should be excluded or minimized include:

a fat. High dietary fat intakes appear to correlate with an increased incidence of bowel, breast, prostate, ovary and endometrial cancers. It has been postulated than fat may act via promoting mechanisms in all these cancers. Reduction of dietary fat may thus decrease the risk of these cancers from fat related causes relatively rapidly. It has been argued that the calories derived from fats usually used in the western diet should be reduced to 20% (and no more than 25%) of the energy intake.[36]

b alcohol. Alcohol appears to act synergistically with cigarettes in increasing the incidence of respiratory tract and lung cancers. Rectal carcinoma has been linked with excessive beer drinking and home brews have been associated with oesophageal carcinomas in certain populations. Alcohol intake should be limited; especially amongst smokers.

c foods contaminated by incomplete fuel combustion, broiled and charred foods.[37] It has been suggested that when barbequing, poultry and fish should be substituted for meat and excess fat should be trimmed from meat prior to cooking. This will minimize the fat dripping and reduce the likelihood of significant quantities of benzopyrene being redeposited on food by smoke. Covering the grill with aluminum foil will reduce flare ups. Precooking will reduce on-the-grill time and any charred food should be discarded.

d nitrates and nitrites. Water, especially in heavily fertilized areas, can be an important source of nitrate. Nitrate, and to a much lesser extent nitrite, occurs naturally in plant food eg beetroot, celery, lettuce, bananas and potatoes. Nitrates and nitrites may also be used in food processing; the minimal amounts of nitrites added to foods to prevent botulism are justifiable on health grounds, while cured or pickled foods may be avoided. Preformed nitrosamines may also be ingested in fish, beer or dairy products. Nitrates are only toxic at levels much higher than that normally occurring in food; it is the conversion of nitrate to nitrite and potentially carcinogenic N-nitrosamines which is of concern. Gastric carcinoma in certain parts of the world appears to be causally related to nitrosamine and nitroso products. Persons at most risk of nitroso related carcinomas are tobacco users with chronic atrophic gastritis, chronic oesophagitis or chronic cystitis. Such persons are advised to reduce their nitrate/nitrite intake and minimize in vivo nitrogenization by the daily ingestion of 1–2 grams of vitamin C.[38,39] It has also been suggested that 400IU of vitamin E each day may be helpful.

e natural food toxins (eg nitrates/nitrites), environmental contaminants and additives. While most concern is frequently

expressed about the health risks associated with food additives, natural food toxins and environmental contaminants constitute a more significant hazard.[40]

Environmental contaminants with carcinogenic potential include fungal contamination eg. aflatoxin, a fumigant for fruit and vegetables (ethylene dibromine), a growth promoter synthetic oestrogen diethylstilbestrol (banned in Australia) and plastic food wrap containing small amounts of non-polymerised vinyl chloride. Protection against environmental contamination depends largely on good food growth, handling and storage. Maintenance of adequate standards in the final food product is largely regulatory by the Model Food Act.[41] Model Food Legislation specifies permissible amounts of potentially noxious substances and ensures that standards are continuously reviewed in the light of advances in the area. Aflatoxins are for example associated with liver cancer and are contaminants of Australian peanuts. Improvements in the storage and handling of grains and nuts can minimize mycotoxin contamination.[42] Although not yet commercially viable, research has shown that aflatoxins can be almost completely destroyed by microwave roasting. In Australia the maximum permissible level of aflatoxin in peanuts and peanut products has currently been set at 15μg/kg.

In Australia the use of food additives is subject to similarly stringent control. Food additives are non-nutritive compounds which are added, usually in small amounts, to food with the intention of improving its taste, texture, flavour or storage properties.[43] Before being approved food additives are subject to toxicological testing. Included in these tests is the Ames test which assesses the mutagenicity of compounds.[44] Tests for carcinogenicity of food additives is performed on animals using high concentrations of the additive. In America the Delaney Clause requires that no additive be permitted should it induce carcinoma in animals or man. In Australia both the types of food additives and the concentrations at which they may be used are specified.

Owing to legislative control in Australia, the only recommendations which one need give a patient seeking to reduce their risk from cancer with regard to environmental contaminants and food additives, is to eat a mixed balanced diet. Eating in moderation is important; the consumption of saccharin in the USA in 1972 was 23mg/day based on a per capita disappearance of the additive; heavy consumers of artificially sweetened soft drinks had an estimated intake of 389 mg/day.

In addition to being a potential source of carcinogens diet can constitute a means of reducing ones cancer risk. The ingestion of high levels of certain nutrients appears to protect against the pathogenesis of cancer. Dietary substances which may reduce the risk of cancer and whose inclusion should be emphasized include[45,7]:

- vitamin A. Rich dietary sources of B-carotene include green leafy and yellow/orange fruit and vegetables. Good sources of retinol include fatty fish and their oils, liver and dairy products; margarine is also a good source due to supplementation. Recommended daily intake for cancer prevention is 12,500 IU. Prolonged ingestion of retinol at levels of 25,000 IU can cause anorexia, headaches, skin changes and muscle bone tenderness; B-carotene has no serious side effects. As the mechanism of action of these components of vitamin A appears to differ, it is desirable that both compounds be regularly ingested.
- vitamin E. While a two and a half times increase of the RDA for vitamin A is recommended, a 10 to 40 fold increase of vitamin E is suggested for cancer prevention. At the recommended cancer risk minimization dose of 200-800IU vitamin E has no toxic effects. It is however worth reminding patients that further increases of vitamin may reduce the efficacy of the immune system (unproven postulate). Good dietary sources of vitamin E include vegetable oils (particularly wheat germ and sunflower seed oil), margarine, eggs, whole grains and cereals. An increased intake of polyunsaturated fats results in an increased requirement for vitamin E.
- vitamin C. At a recommended dose of 1.0 gram of ascorbic acid per day the RDA for this vitamin is exceeded 17 fold. At this level the side effects of vitamin C supplementation are negligible. Less conservative approaches to cancer prevention may recommend vitamin C supplementation up to the level of bowel tolerance. Good dietary sources of vitamin C are fruit and vegetables. Smokers, women on oral contraceptives and diabetes require increased levels of this vitamin.
- selenium. Conservative recommendation of selenium as a cancer risk reducing agent suggest a daily intake of 50–200μg. As this mineral has significant toxicity, it, unlike vitamins A, E and C is not available as an over-the-counter supplement. High intakes of vitamin E reduce the requirement for selenium. Dietary sources of selenium are fish, meat and cereals. Food selenium content varies with soil content; New Zealand soil, particularly the south island, is depleted of this mineral.
- fibre. A daily intake of 35 grams of fibre is recommended. Fibre is not a homogenous substance. Cellulose is non-digestible and therefore remains in the intestinal lumen and may influence intestinal flora and bacterial metabolism, pentose increases stool softness and bulk thereby also reducing gut transit time while lignin binds bile acids. All these factors may reduce the risk of bowel cancer by reducing the contact time between mucosa and carcinogens within the bowel lumen. Foods rich in fibre include beans and split peas (4gm/serve), almonds or peanuts (3gm/10nuts) and dried fruit eg raisins (6gm/cup).

Summary of dietary recommendations

In order to minimize their patient's risk of cancer by dietary means, chiropractors may make the following recommendations:

- lose excess weight and attempt to maintain an ideal body weight (approximate Ideal Body Weight (Kg) = Height (cm) − 100)

- eat about 35 grams of fibre each day. A diet rich in bran cereals, whole grains, nuts, dried fruit and vegetables is required. Rapid conversion from a low to high fibre diet may cause bowel distention and gas production. Gradual increases of fibre intake are preferable as this reduces patient symptoms and facilitates intestinal adaptation of mineral absorption.
- reduce fat intake to about 30% of caloric intake. Calculation of permissible fat intake in grams/day is determined by dividing 37 (the energy/kilojoule concentration in one gram of fat) into 30% of the patient's daily energy allowance. Limit butter, margarine, oils and lard; select low fat dairy products, remove all visible fat from meat and eat fish and free range poultry in preference to red meat. Substitution of vegetable for animal proteins reduces fat and increases fibre and vitamins C, E and A (β-carotene).
- eat more vegetables and fruit. At least two serves each day of green leafy or yellow vegetables and yellow/orange fruit is suggested. Cruciferous vegetables (cauliflower, cabbage, brussel sprouts) and asparagus are also particularly well suited to a cancer prevention diet. They are good sources of fibre, carotene and vitamin C; they also contain indoles which may exert a further protective effect against breast, stomach and colorectal carcinomas.[46]
- avoid excess alcohol and stop smoking
- avoid eating charred, burnt, smoked, cured or pickled foods.
- avoid excessive ingestion of any particular foodsubstance eg peanuts or peanut butter.

All patients should be advised to adhere to the above regime. Patients who are deemed to be at particular risk of cancer may require additional protection. While further evidence is desirable there is some justification for conservative prescribing of the following supplements:

- vitamin C 1–2 grams per day. Smokers and persons at increase risk of in vivo nitrosamine production should be placed at the 2 gram level.
- vitamin E 200–800 IU; but not more than 800 IU.
- vitamin A 12,500 IU. Intakes of retinol may be reduced where increased ß-carotene is available.

Clinical intervention for cancer prevention

While diet has been emphasized as a mode of cancer risk reduction in this paper, clinical intervention for cancer prevention requires a more holistic approach. Chiropractors functioning at the primary level of health care may include nutritional measures in an overall cancer prevention program. Such a program should take cognisance of other variables which may enhance and other strategies which may reduce the patient's likelihood of developing and/or dying from cancer. Variables which increase an individual's risk of cancer include:

- a family history of the disease;
- specific conditions which predispose to certain cancers, eg. persons with ulcerative colitis are at increased risk of developing colonic carcinoma; mammary dysplasia increases the risk of breast cancer;
- age. While certain cancers peak at various agegroups, the overall risk of cancer increases with advancing age;
- smoking; and
- exposure to ionizing radiation.

Strategies which may reduce an individual's risk of developing cancer include:

- avoiding known carcinogens (ie initiators and promotors). Food selection and preparation is important; and
- impeding carcinogenesis, eg. neutralizing/inactivating promotors. Vitamin supplementation is potentially useful.

Active screening for the early detection of neoplasia is also important.[47,48,49] In the case of breast cancer screening starts by preparing the patient to perform monthly breast self-examination at age 20. A baseline mammography should be performed between the ages of 35–40 years. After this time physicial breast examinations should be performed annually with mammography every second year to the age of 50 years. Thereafter, in addition to monthly self-examination, physician examination and mammography should be performed at yearly intervals. Early detection improves the prognosis of breast cancer.

Screening for colorectal carcinoma is also recommended. All persons over the age of 50 years should have annual stool guaiac tests for occult blood. Reputable sources also recommend sigmoidoscopy, a more invasive screening technique, at 3 to 5 yearly intervals in this age group. Invasive screening starts at a younger age in the case of cervical carcinoma. Pap smears are recommended at 3 yearly intervals in sexually active women from 16 years of age.

Intervention which aims to achieve cancer preventions needs to be initiated as early as possible and requires lifelong committment. Such committment is only possible when the patient is actively involved in formulating the intervention contract.[50]

Concluding remarks

1 Diet does appear to be involved in the pathogenesis of a number of prevalent carcinomas.
2 Dietary avoidance of potential carcinogens is desireable but not always possible. While legislative control can largely ensure reasonable food safety with regard to additives and certain environmental contaminants, it lacks the ability to prevent excessive consumption of particular foods by individuals. Legislative control is also powerless to enforce healthy home cooking and storage methods.
3 Moderate nutritional supplementation, while requiring further investigation, does appear to offer a potential means of cancer risk reduction.

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