Review Article

# Bioactive phytochemicals in Indian foods and their potential in health promotion and disease prevention

Bs Narasinga Rao, BSc (Hons), PhD

*National Institute of Nutrition, Hyderabad, India*

Besides being a source of nutrients, foods, particularly plant foods, are a rich source of bioactive phytochemicals or bionutrients. Studies carried out during the past 2–3 decades have shown that these phytochemicals have an important role in preventing chronic diseases like cancer, diabetes, coronary heart disease and hyper-cholesterolaemia. The major classes of phytochemicals with disease-preventing functions are dietary fibre, antioxidants, detoxifying agents, immunity-potentiating agents and neuropharmacological agents. Each class of these functional agents consists of a wide range of chemicals with differing potency. For example, antioxidant function is exhibited by some nutrients, such as vitamin E, vitamin C and provitamin A. Other phytochemicals that have antioxidant properties are carotenoids, phenolic compounds, flavonoids and isothiocyanates. Some of these phytochemicals have more than one function. Foods rich in these chemicals and exhibiting disease-protecting potential are called functional foods. Indian habitual diets, which are based predominantly on plant foods like cereals, pulses, oils and spices, are all good sources of these classes of phytochemicals, particularly dietary fibre, vitamin E, carotenoids and phenolic compounds. There is, however, much scope for further systematic research in screening Indian foods and diets for these phytochemicals and assessing their potential in protecting against chronic diseases.

**Key Words: antioxidants, bionutrients, diabetes, functional foods, phytochemicals, Indian foods, spices.**

## Introduction

Certain foods are essential for maintaining good nutrition and health in humans. Studies conducted since the mid-nineteenth century have been centred on the identification of essential nutrients in foods, and have included the study of their metabolism and functions. Studies have also been focused on dietary deficiency of these nutrients and their clinical and functional consequences. Food technology and food processing have focused on conserving these nutrients in processed foods. Much research has also been carried out on the human requirement of these nutrients and strategies to combat nutritional deficiency diseases among underprivileged population groups. A list of nutrients currently known to be present in foods and that are essential for man are given in Table 1.

While the main focus of attention before the 1980s was on the nutrient content of foods, there were also studies on some antinutritional and toxic factors that are present in various foods (Table 1). These non-nutrient components included antitrypsins, which interfere with dietary protein utilization, phytates and tannins, which interfere with iron absorption, and oxalates, which inhibit dietary calcium absorption. Other non-nutrient phytochemicals that were the focus of attention were food toxins. These included toxic amino acids, ßN oxalyl L αß diamino propionic acid (ODPA) in lathyrus (*Lathyrus sativus*) seeds that lead to lathyrism, and goitrogens, such as thiocyanates and isothiocyanates, that interfere with iodine utilisation for the synthesis of thyroid hormones, leading to goitre or iodine deficiency disorders. During 1960s and 1970s, attention was primarily focused on natural and environmentally-derived toxins such as aflatoxin, pesticide residues and heavy metal contamination. There were also concerns about the role of diet in chronic diseases like diabetes, atherosclerosis, cardiovascular diseases and obesity, due to excessive consumption of nutrients like energy, fat and some fat-soluble nutrients.

## Bioactive phytochemicals and functional foods

It is well recognised that foods are the main sources of nutrients used to meet our nutritional needs. However, foods, particularly those of plant origin, contain a wide range of non-nutrient phytochemicals that are elaborated by plants for their own defence and for other biological functions. When man ingests these plant foods to meet his nutritional needs, he also ingests a wide variety of these non-nutrient phytochemicals.

**Correspondence address:** Dr BS Nasaringa Rao, 1-2-62/3, 11/4 M Block, Kakateeyanagar, Habsiguda, Hyderabad 500 007, India.
Tel: +91 040 717 3344; Fax: +91 040 701 9074
Email: bnmohan@hd2.dot.net.in
Accepted 12 February 2002

Many plants and herbs are considered to have medicinal value, as described in the ancient Indian medical system (Ayurveda) and also in folk medicine. They have been in use as part of home remedies for several common ailments.[1] These phytochemicals present in commonly consumed plant foods are normally non-toxic and have the potential for preventing chronic diseases.

There have been several attempts during the early decades of the twentieth century by leading organic chemists (such as the late Professor Sheshadri, and his associates) to study the active principles of several medicinal plants in use in India.[1] Such studies had focused attention mostly on their detection, isolation and characterisation. The compounds included flavones, flavonoids and alkaloids that were present in local plant material. Some studies were also carried out concurrently to determine their pharmacological properties. There were, however, no concerted efforts to coordinate the chemical and biological studies on these phytochemicals to identify their disease-preventing potential, if any.

For the last two to three decades, however, there has been a surge of interest in plant foods as a source of phytochemicals that may have a useful role in the prevention of chronic diseases such as cancer, diabetes, cardiovascular disease, cataract and gallstone.[2] Such an association between consumption of certain foods and low prevalence of non-communicable disease was derived initially from epidemiological observations. Foods that have disease-preventing potential are designated 'functional foods'. Functional foods are foods that provide health benefits beyond basic nutrition. 'The appeal of functional foods lies in their potential to lower the incidence of diet related diseases. And, with a rapidly ageing world population, the cost of national health bills.'[3]

Health claims for certain foods are permitted by the health, food and nutrition authorities of several countries, including USA, Europe, Australia and Japan. Japan has taken a leading role in this regard, where several functional foods, both natural and processed, are marketed. In Japan, functional foods are called FOSHU (foods for specific health use) and are defined as foods derived from naturally occurring substances (not a capsule or powder) that can regulate a particular body process when consumed as part of the daily diet.[3] The FOSHU are classified into five categories, namely:

(i) enhancers of the body's immune system;
(ii) preventers of diabetes and heart diseases;
(iii) hypocholesterolaemic agents;
(iv) promoters of digestion and absorption; and
(v) retardants of ageing.

The phytochemicals present in functional foods that are responsible for preventing disease and promoting health have been studied extensively to establish their efficacy and to understand the underlying mechanism of their action. Such studies have included identification and isolation of the chemical components, establishment of their biological potency both by *in vitro* and *in vivo* studies in experimental animals and through epidemiological and clinical-case control studies in man. These studies have been very extensive in the case of plant foods and the phytochemicals present therein in relation to their cancer-preventing potential. Disease-preventing phytochemicals present in plant foods are shown in Table 2. Properties of functional foods are shared by several phytochemicals present in plant foods, as shown in Table 3. It is interesting to note that several nutrients like vitamin E (tocopherols), provitamin A (β-carotene), ascorbic acid and selenium also have disease-preventing and health-promoting potentials, just like phytochemicals (Table 3). Research on the health-promoting and disease-preventing potential of phytochemicals in foods have grown so much in recent years that this branch of research has been named bionutrition.[4]

**Table 1.** Nutrients and antinutrients in foods

| Category | Nutrient/antinutrient |
|---|---|
| Macronutrients | Energy, protein, fat |
| Minerals | Calcium, phosphorus, magnesium, chloride, iron, sodium, potassium |
| Micronutrients | |
| Vitamins | Vitamin A, vitamin D, vitamin E, vitamin K, essential fatty acids, thiamine, riboflavin, nicotinic acid, pyridoxine, folate, vitamin $B_{12}$, ascorbic acid, biotin, inositol, choline, paraaminobenzoic acid |
| Trace elements | Zinc, copper, cobalt, chromium, molybdenum, vanadium, selenium, iodine, fluoride, silicon |
| Antinutrients | |
| Antitrypsin | Protein |
| Oxalate | Calcium |
| Phytate | Calcium, iron |
| Polyphenols | Iron, protein |
| Goitrogens | Iodine |
| Toxic factors | |
| Oxdiapro | Lathyrism |
| Argimone | Spedenic Dropsy |
| Mycotoxins | Liver injury |

**Table 2.** Disease-preventing phytochemicals in plant foods

| Specific foods/food groups | Main health promoting chemical | Useful in the prevention of |
|---|---|---|
| Whole cereal, grains | Dietary fibre, tocopherols† | Cancer, diabetes, cardiovascular disease, hypercholesterolaemia |
| Vegetables, fat | α-linoleic acid, linoleic acid, tocopherols,† sterols | Cancer, diabetes, cardiovascular disease, hypercholesterolaemia |
| Palm oil | Tocopherols,† tocotrienols,† carotenoids | Cancer, heart diseases, atherosclerosis, cataract, pulmonary diseases, muscle injury |
| Yellow or green leafy vegetables, yellow fruits | Carotenes,† ascorbic acid,† dietary fibre, omega-3 fatty acids, pectins | Cancer, heart diseases, atherosclerosis, cataract, pulmonary diseases, muscle injury |
| Rice bran oil | Sterols, PUFA | Hypercholesterolaemia, diabetes, cardiovascular disease |
| Linseed oil, fish oil | Omega-3 fatty acid | Hypercholesterolaemia, diabetes, cardiovascular disease |
| Spices, fenugreek seeds, turmeric | Gums, curcumin, eugenol, capsicin | Cancer, cardiovascular disease, detoxification of drugs and toxins |

†Antioxidant. PUFA, polyunsaturated fatty acid.

**Table 3.** Bioactive phytochemicals in foods

| Classification | Main groups of compounds | Biological function |
|---|---|---|
| NSA | Cellulose, hemicellulose, gums, mucilages, pectins, lignins | Water holding capacity, delay in nutrient absorption, binding toxins and bile acids |
| Antioxidants | Polyphenolic compounds, flavonoids, carotenoids, tocopherols, ascorbic acid, anthocyanine, phenolic indoles | Oxygen free radical quenching, inhibition of lipid peroxidation |
| Detoxifying agents | Reductive acids, tocopherols, phenols, indoles, aromatic isothiocyanates, coumarins, flavones, diterpenes, carotenoids, retinoids, cyanates, phytosterols, methyl xanthines, protease inhibitors | Inhibitors of procarcinogen activation, inducers of drug-metabolising enzymes, binding of carcinogens, inhibitors of tumourogenesis |
| Others | Alkaloids, volatile flavour compounds, biogenic amines, terpenoids and other isoprenoid compounds | Neuropharmacological agents, anti-oxidants, cancer chemoprevention |

NSA, non-starch polysaccharides.

Although these biologically active phytochemicals have been reported to be present in a wide range of plant foods, their efficacy in disease prevention has been clearly established only in the case of some. The potency of a given functional food in preventing a disease is the sum of the bioactivities of all phytochemicals present in that food with a common property. Whether there is synergism between these chemicals, they are additive or only one or two components account for most of the functions of the food are questions that remain unanswered. These phytochemicals are reported to prevent disease mainly through their functions as antioxidants, detoxifiers, neuropharmacological and immuno-potentiating agents, and as a source of dietary fibre (non-starch polysaccharide, NSP).

**Dietary fibre or non-starch polysaccharides**

Most plant foods in their native state contain indigestible residues that used to be classified as crude fibre, but are currently classified as dietary fibre (DF) and also as NSP. Dietary fibre is not a single entity but consists of a wide range of complex polysaccharides such as cellulose, gums, mucilages, hemicellulose and lignins with different chemical, physiochemical and physiological properties (Table 4). The Central Food Technological Research Institute (CFTRI), Mysore, India has studied the properties of these NSP in Indian foods in relation to their role in conferring desirable functional properties on processed foods.[5] These dietary fibres (NSP) in foods have been shown to be useful in reducing blood glucose levels in diabetes, in reducing blood cholesterol levels for treatment of cardiovascular disease and also in preventing bowel cancer.[6,7] The disease-preventing potential of DF will depend upon the proportion and actual

**Table 4.** Components of dietary fibre

| Major class | Property | Chemical nature |
|---|---|---|
| Cellulose | Water insoluble | Linear 1–4 β-glycans |
| Non-cellulose polysaccharides | | |
| Pectin | Water soluble | Galacturonic acid, neutral sugars |
| Hemicellulose | Water insoluble | Xylose, arabinose, galactose, mannose |
| Gums | Water soluble | Xylose, arabinose, rhaminose |
| Mucilages | Water soluble | Galactose, galacturonic acid, rhaminose |
| Lignin | Water insoluble | Polymer of hydroxyphenylpropane derivatives |

**Table 5.** Some properties of dietary fibre and their health consequences

| Function | Health consequence |
|---|---|
| Water absorbing and bulking property | Energy diluent to formulate low calorie diets |
| Increased transit time of food in the gut | Reduced risk of inflammatory bowel disease |
| Bile acid and steroid binding | Hypocholesterolemic activity and reducing the risk of cardiovascular diseases |
| Retardation of carbohydrate absorption and impaired glucose tolerance | Management of certain type of diabetes |
| Binding of toxins | As a detoxifying agent |
| Binding of divalent cations | Reduced bioavailability of Ca, Mg, Zn, Fe |

quantities of different polysaccharide components present in a given food, and these have been reviewed by this author.[8] Although many plant foods contain several NSP components, all of them may not exert the desired health benefit to the same degree. Diets based only on foods containing a specific type of NSP at a reasonably high intake level of 30–50 g/day can exert the expected beneficial effect. For example, fenugreek (*Trigonella sativum*) seeds, which contain 40% gum, can have a desirable beneficial effect on controlling blood glucose levels only at an intake of 80–100 g/day.[9,10]

Dietary fibre components exert their beneficial effects mostly by way of their swelling properties, and by increasing transit time in the small intestine (Table 5). Consequently, they reduce the rate of release of glucose and its absorption, thus helping in the management of certain types of diabetes (e.g., non-insulin-dependent diabetes mellitus). DF components also bind bile salts, thereby promoting cholesterol excretion from the body and thus reducing blood cholesterol levels, and food toxins in the gut to reduce their toxicity. They can also have some adverse nutritional effects by binding dietary calcium, magnesium, zinc and iron, thereby reducing their bioavailability.

The second mechanism by which dietary fibre exerts its beneficial effects is through undergoing fermentation in the large intestine (colon) and producing short-chain fatty acids such as butyrate, propionate and acetate. Butyrate helps in the regeneration of colon mucosal cells by serving as a source of energy, thereby reducing the risk of colon cancer and inflammatory bowel disease. The short-chain fatty acids produced are absorbed (especially propionate and acetate) into splenic circulation and transported to the liver where they are known to inhibit cholesterol synthesis by hepatocytes and also glucose release from the liver, thus contributing partly to the hypocholesterolaemic and hypoglycaemic effects of dietary fibre. While the soluble fibres are completely fermented, the insoluble fibres are only partially fermented.

***Dietary fibre intake in India***

It is generally recommended that the daily intake of dietary fibre should be around 40 g for an adult. In habitual Indian diets, being based predominantly on unrefined cereals and plant foods, this level of dietary fibre intake is easily achieved. The dietary fibre contents of several Indian foods have been determined[8,9] and are shown in Table 6. Dietary fibre intake in India by different socioeconomic groups varies from 60 to 70 g/day, depending upon the type of cereal consumed (Table 7). Dietary fibre intakes in wheat- or millet-based diets are generally higher than in a rice-based diet. Nearly 80% of the fibre content of the diets consumed in India, particularly among the low-income group, is contributed by cereals. Although the fibre intake of Indians through their cereal-based diets is quite adequate according to the currently recommended desirable intakes, the exact composition of the individual fibre fractions in most Indian foods has not been determined. However, some of the fractions of DF, including soluble and insoluble NSP, have been determined in cereals and pulses, the major sources of DF (Table 8). The soluble dietary fibre content of foods is reported to vary widely:[6] cereals, 8–20%; legumes, 20–50%; vegetables, 25–67%; and fruits 20–65%. Diets of high-income groups, which include fruits, vegetables and legumes, will therefore contain more soluble NSP than diets of the poor, although the total NSP content of the diets of the two groups may be the same.

**Table 6.** Dietary fibre content of some common Indian foods

| Foodstuff | Energy (kCal) | Crude fibre (g) | Dietary fibre (g) |
|---|---|---|---|
| Cereals and millets | | | |
| Rice | 345 | 0.2 | 4.1–8.3 |
| Wheat | 346 | 1.2 | 11.4–17.2 |
| Sorghum | 349 | 1.6 | 9.7–14.3 |
| Bajra | 361 | 1.6 | 11.8–20.3 |
| Ragi | 328 | 3.6 | 11.5–18.6 |
| Pulses and legumes | | | |
| Greengram (whole) | 334 | 4.1 | 15.2 |
| Greengram (dhal) | 348 | 0.8 | 13.5 |
| Blackgram (dhal) | 347 | 0.9 | 14.3 |
| Redgram (dhal) | 335 | 1.5 | 14.1 |
| Bengalgram (whole) | 360 | 3.9 | 26.6 |
| Bengalgram (dhal) | 372 | 1.2 | 13.6 |
| Nuts and oilseeds | | | |
| Groundnut | 567 | 3.1 | 6.1 |
| Coconut (dry) | 662 | 6.6 | 8.9 |
| Roots and tubers | | | |
| Sweet potato | 120 | 0.8 | 7.3 |
| Potato | 97 | 0.4 | 4.0 |
| Yam | 79 | 0.8 | 5.3 |
| Fruits | | | |
| Banana | 116 | 0.4 | 2.5 |
| Mango | 74 | 0.7 | 2.3 |
| Vegetables | | | |
| Amaranth | 45 | 1.0 | 3.4 |
| Palak | 26 | 0.6 | 5.0 |
| Brinjal | 24 | 1.3 | 2.0 |
| Ridge gourd | 17 | 0.5 | 5.7 |
| Snake gourd | 18 | 0.8 | 1.8 |
| Bottle gourd | 12 | 0.6 | 2.8 |
| Yellow Pumpkin | 23 | 0.7 | 0.5 |

**Table 7.** Dietary fibre intake by urban and rural groups in India†

| Group | Energy (kCal) | Dietary fibre‡ |
|---|---|---|
| Urban | | |
| High income | 2604 | 59.4 |
| Middle income | 2230 | 61.5 |
| Low income | 2008 | 67.7 |
| Rural | | |
| Average for different income groups | 2272 | 72.7 |

†Average intake per consumption unit. ‡From a cereal-based diet.[8]

**Antioxidants: their role in disease prevention**

It is currently believed that reactive oxygen species (ROS) have an important role in the aetiology of several non-communicable diseases. Oxidants and free radicals such as singlet molecular oxygen ($^{-}O_2$), superoxide ($^{-}O$), hydroxyl (OH) peroxide (O-O-H) and lipid peroxides (LOO) are known to cause tissue damage.[11,12] Such free radicals also include nitrous oxide radicals that are generated in the gastrointestinal tract. Tissue damage caused by free radicals, when it becomes cumulative, is considered to play an important role in the pathogenesis of several degenerative diseases, for example, cancer, cataract, coronary heart disease, dementia, diabetes mellitus, rheumatic arthritis, muscular degeneration, pulmonary dysfunction and radiation sickness. Lipid peroxidation of membrane lipids, circulating lipoprotein lipids (including cholesterol), oxidation damage of cellular proteins and DNA, and lens proteins in the retina are all considered mechanisms by which the oxygen free radicals and peroxides lead to these diseases. These oxidants and free radical species are generated in cells during utilisation of oxygen, which is essential for life's sustenance, and they can also be derived from external sources.

There are, however, protective mechanisms within the body that protect against dangerous oxygen free radical species generated *in situ*, and those derived from external sources. Superoxide dismutase (SOD) can convert the $O_2$

**Table 8.** Dietary fibre fractions in cereals and pulses consumed in India

| Foodstuff | Total fibre (g/100 g) | NSP (g/100 g) Total | NSP (g/100 g) Water soluble | Cellulose (g/100 g) | Lignin (g/100 g) |
|---|---|---|---|---|---|
| Cereals and millets | | | | | |
| Rice | 8.3 | 4.1 | 0.9 | 2.7 | 1.5 |
| Wheat | 17.2 | 10.6 | 2.5 | 5.5 | 1.2 |
| Sorghum | 14.2 | 7.6 | 1.3 | 3.5 | 3.1 |
| Bajra | 20.5 | 12.5 | 2.4 | 6.2 | 1.8 |
| Ragi | 18.6 | 6.1 | 0.9 | 4.6 | 7.9 |
| Pulses and legumes | | | | | |
| Redgram | | | | | |
| Whole | 20.3 | 10.2 | 1.4 | 7.3 | 2.9 |
| Decorticated | 13.4 | 10.3 | 1.7 | 2.1 | 1.0 |
| Bengal gram | | | | | |
| Whole | 25.6 | 9.1 | 3.3 | 13.7 | 2.8 |
| Decorticated | 13.4 | 7.1 | 1.3 | 5.5 | 0.9 |
| Black gram | | | | | |
| Whole | 19.5 | 10.7 | 2.4 | 5.0 | 3.8 |
| Decorticated | 13.6 | 9.5 | 2.6 | 2.9 | 1.2 |
| Green gram | | | | | |
| Whole | 15.2 | 8.5 | 1.5 | 4.6 | 2.2 |
| Decorticated | 13.2 | 8.6 | 2.9 | 3.7 | 1.0 |

NSP, non-starch polysaccharides.

radical to hydrogen peroxide, which can be further destroyed by catalase. Glutathione reductase, for which riboflavin is the coenzyme, is also a part of the *in vivo* defence system against oxidation damage in the cells. Selenium too plays a role in this system through glutathione peroxidase. This *in vivo* system of detoxifying oxygen free radicals may not be capable of neutralising all of the free radicals produced in the body as well as those derived from the environment, and there is therefore a need for an external source of antioxidants to neutralise the free radical load in the body.

A large number of antioxidants, both nutritive and non-nutritive, occur in foods. Besides β-carotene, vitamin C and vitamin E (which are nutrients), a number of carotenoids, phenols and flavonoids also occur naturally in foods and can act as antioxidants. Most plant foods contain phenols and flavonoids. Green leafy vegetables, fruits and yellow vegetables are particularly rich in carotenoids, flavonoids and vitamin C. Vitamin C and vitamin E prevent formation of nitrosamine, which is carcinogenic. Vitamin E also protects selenium against reduction and protects polyunsaturated fatty acids (PUFA) in the membrane against oxidative damage. Green leafy vegetables and fruits like papaya can be an inexpensive source of carotenoids that do not have provitamin A activity but possess antioxidant properties. Vitamin E is present in vegetable oils and the germ portion of cereals. Palm oil is a rich source of both tocopherols and tocotrienols. Other edible oils rich in antioxidants are rice bran oil and sesame oil. Rice bran oil contains oryzanol, which can act as an antioxidant, and is rich in plant sterols that, along with oryzanol, effectively lower blood cholesterol. Sesame oil contains sesaminol, which is a powerful antioxidant. Spices are also rich in phenolic compounds that have been shown to act as antioxidants. Turmeric (*Curcuma domestica*), which is widely used in Indian cooking, contains a yellow colouring principle, curcumin, which is a powerful antioxidant and can offer protection against cancer. Foods popularly advocated as being rich sources of vitamin C, such as amla (*Emblica officinalis*) and guava (*Psidium guajava*), can also be rich and inexpensive sources of antioxidants. Besides being rich in vitamin C, these two fruits also contain phenolic compounds, including flavonoids.

The total antioxidant potential of a food or diet can be determined by its capacity to prevent lipid peroxidation in an *in vitro* system. Green leafy vegetables are good sources of antioxidants, contributed by their content of carotenoids, flavonoids and tocopherols. However, the potency of antioxidants present in foods *in vivo* will depend not only on their levels in the foods but also on their bioavailability, that is, the extent to which the active forms of antioxidants are released from the food and absorbed through the gut. Some flavonoids and phenolic antioxidants are rather poorly absorbed; they often form insoluble complexes with metals such as iron. Their antioxidant potency will also depend on the oxidant level in the food, for example, high PUFA content, which is prone to lipid peroxide formation, can reduce the antioxidant potency of a food.

In the past, lipid peroxide levels were measured in terms of thiobarbituric acid-reactive substances, which now is considered rather non-specific. Currently, new methods have been developed for separating lipid peroxides from their

breakdown products before their levels are estimated. Elegant methods employing high-performance liquid chromatography (HPLC), gas chromatography and mass spectrometry (GCMS), chemiluminescence $^{13}$C-nuclear magnetic resonance and enzymatic methods have been developed for assessing lipid peroxidation levels in body fluids and tissues.

The total antioxidant ability of foods can also be tested *in vivo* by measuring the effect of their ingestion on biological fluids.[13,14] Such tests include the total peroxyl radical trapping parameters assay and the ferric reducing antioxidant power assay. Besides antioxidant ability, the pro-oxidant stress (i.e. ROS) should also be tested to assess the pro-oxidant and antioxidant balance. ROS can be determined by chemiluminescence or electroparamagnetic spectroscopy, or the ROS can be 'trapped' and changes in or destruction of the trapping agent can be measured.

The beneficial influence of antioxidants and antioxidant-rich foods on the prevention of diseases such as cancer has been studied extensively through *in vitro* cell culture, animal and human studies. The association between the intake of dietary antioxidants and protection against non-communicable diseases in humans (cancer, cardiovascular diseases, cataract) have been largely based on epidemiological studies. However, there have been more systematic human studies involving nutrient antioxidants like vitamin A β-carotene, vitamin C and selenium in the prevention and treatment of cancer caused by several carcinogenic agents. Based on these studies, the amount of these antioxidants recommended for cancer prevention is 2–20 times the recommended dietary allowance (RDA) for their vitamin function (Table 9).

There have been several recent epidemiological studies that implicate dietary antioxidant phytochemicals like carotenoids,[15,16] phenolic compounds[17] and flavonoids[18] as protective agents against cancer and cardiovascular disease. Zeigler *et al.*[19] have shown that intake of carotenoids besides β-carotene, like lutein, zeaxanthin and lycopene, through fruits and vegetables resulted in elevated levels of blood carotenoids. This, in turn, was associated with a reduced risk of lung cancer. Hertog *et al.*[20] have shown in an epidemiological study in the Netherlands that regular intake of flavonoids (quercetin and kaemferol) by the elderly, through vegetables and fruit (26 mg/day), reduces the risk of death from coronary heart disease. In that study, flavonoids consumed in fruits and vegetables were determined by HPLC. These flavonoids act by scavenging superoxide anions, singlet oxygens and lipid peroxyradicals, and through sequestering metal ions that promote oxyradical formation. Quercetin, the major flavonoid, inhibits oxidation and cytotoxicity of low-density lipoprotein. Flavonoids also inhibit cyclooxygenase, leading to lower platelet aggregation and reduced thrombotic tendencies.

**Table 9.** Recommendations for cancer prevention

| Antioxidant | RDA | Recommended intake as antioxidant | Possible toxicity levels | Factors possibly increasing requirements |
|---|---|---|---|---|
| Vitamin E | 7–14 mg | 133–533 mg | Negligible at less than 1200IU (800mg) | High polyunsaturated fat intake, smog, smoke |
| Vitamin A† | 5000 IU (1.5 mg) | 12 500 IU (3.4 mg) | Limited (chronic intakes above 25 000 IU, acute intakes of 30 000 IU or over) | Smoking |
| Vitamin C | 60 mg | 1000 mg | Negligible at 1–2 g | Emotional and environmental stress, oral contraceptives, smoking |
| Selenium | None | 50–200 μg | Significant potential toxicity at over 200 μg | Ageing, high polyunsaturated fat intake, smog, heavy metal contamination |

†1 mg vitamin A = 4 mg β-carotene.

**Table 10.** Total antioxidant activity of some Indian foods

| Food item | μg α-tocopherol equivalent/100 g fresh weight |
|---|---|
| Rice (*Oryza sativa*) | 80–102 |
| Whole wheat (*Tritium aestivum*) | 220–500 |
| Spinach (*Spinacia oleracea*) | 750–890 |
| Amaranth (*Amaranthus gangeticus*) | 620–810 |
| Coriander leaves (*Coriander sativum*) | 610–750 |
| Fenugreek leaves (*Trigonella sativum*) | 520–690 |
| Gogu (*Hibiscus cannabinus*) | 700–810 |
| Mint (*Mentha spicata*) | 630–720 |
| Ponnuganti (*Alternanthera sessitis*) | 500–630 |
| Ambati chukka (*Rumex vesicarius*) | 530–610 |

Taken from the Annual Report, 1997-1998, National Institute of Nutrition, Hyderabad, India.

In another study by Frankel *et al.*,[21] it was reported that phenolic substances in red wine inhibit oxidation of human low-density lipoproteins, providing an explanation for the low prevalence of coronary heart disease (CHD) in some regions of France in spite of a high intake of saturated fat (a known risk factor for CHD). This anomaly, called the French Paradox, is attributed to regular consumption of red wine rich in phenolic substances.

Epidemiological studies designed to correlate the intake of antioxidant phytochemicals with a low prevalence of cancer or heart disease have their limitations as several other environmental, dietary and genetic factors can influence these diseases.

**Table 11.** β-Carotene and ascorbic acid-rich foods available in India

| Food | β-Carotene (μg/100 g) | Ascorbic acid (mg/100 g) |
|---|---|---|
| Amaranth | 5520 | 99 |
| Agathi | 5400 | 169 |
| Coriander leaves | 6918 | 135 |
| Curry leaves | 7560 | 4 |
| Mint | 1620 | 27 |
| Mustard leaves | 2620 | 33 |
| Spinach (Bachali) | 5580 | 28 |
| Rape leaves | 1330 | 65 |
| Ponnaganti | 1926 | 17 |
| Fenugreek leaves | 2340 | 52 |
| Drumstick leaves | 6700 | 220 |
| Cluster beans | 198 | 49 |
| Chillis (green) | 1007 | 111 |
| Carrots | 6460 | 168 |
| Amla | – | 600 |
| Lime | – | 63 |
| Tomato (ripe) | 590 | 27 |
| Papaya (ripe) | 880 | 57 |
| Mango (ripe) | 1990 | 16 |
| Guava (country) | – | 212 |
| Lemon | – | 39 |

It is a question of balance between pro-cancer or pro-CHD factors like free radicals, generated by pro-oxidants, against the level of antioxidant intake. However, the effect of consuming antioxidant-containing foods on lipid peroxide levels, which are indicators of cell damage leading to these non-communicable diseases (NCD), can be directly determined, as described above.

***Antioxidant contents of Indian foods and their dietary intake***

The antioxidant potentials of some Indian foods have been determined by the *in vitro* method, based on their effect on preventing lipid peroxidation (Table 10). As far as the individual antioxidant contents of Indian foods are concerned, we have sufficient data only on the content of vitamin antioxidants in our foods (ascorbic acid, carotene, tocopherol

**Table 13.** Thiocyanate and phenolic contents of Indian foods

| Food group | SCN (mg/100 g) | Phenolics (g/100 g) |
|---|---|---|
| Cereals and millets | 0.2–0.9 | 0.1–2.9 |
| Pulses | 0.3–0.8 | 0.6–5.6 |
| Vegetables (non-leafy) | 0.5–5.7 | 0.2–3.8 |
| Leafy vegetables | 0.3–10.1† | 1.4–3.7 |
| Roots and tubers | 0.2–18.6‡ | 0.6–2.7 |
| Oilseeds and other seeds | 0.8–16.2§ | 0.7–5.6 |
| Spices and condiments | 0.3–8.5 | 0.2–3.7 |
| Fruits | 0.2–1.6 | 0.9–4.4 |

†Cabbage; ‡tapioca; §mustard. Leela Mahesh Rao D. Studies on Iodine metabolism. Phd Thesis, Osmania University, Hyderabad, India, 1993: 42-43.

**Table 14.** Daily intake polyphenolic compounds (per CU*) in Indian rural diets

| Regional diet | Catechin equivalents (mg) | |
|---|---|---|
| | Low income | Very low income |
| Rice based | 2230 | 1495 |
| Wheat based | 2474 | 2148 |
| Sorghum based | 2318 | 1507 |

*Consumption unit of an adult reference man.

**Table 12.** Average tocopherol content of foods and average daily intake of tocopherol in India

| Food | Average content (mg/100 g) | Daily intake (mg/CU*) | Contribution of food (%) |
|---|---|---|---|
| Cereals and millets | 1.2 | 5.57 | 37.7 |
| Pulses and legumes | 3.8 | 1.29 | 8.7 |
| Leafy vegetables | 2.0 | 0.34 | 2.3 |
| Other vegetables | 1.0 | 0.48 | 3.3 |
| Roots and tubers | 0.1 | 0.05 | 0.3 |
| Fruits | 0.5 | 0.06 | 0.4 |
| Nuts and oilseeds | 16.0 | 1.35 | 9.5 |
| Oils and fats | 47.5 | 4.75 | 32.2 |
| Milk | 0.1 | 0.72 | 4.9 |
| Animal foods | 0.1 | 0.16 | 1.1 |
| Total | – | 14.77 | 100.0 |

* Consumption unit of an adult reference man.

**Table 15.** Hypocholesterolaemic and antioxidant potentials of some edible oils

| Edible oils | Hypocholesterolemic agents (%) | | | Antioxidant agents (%) | | | |
|---|---|---|---|---|---|---|---|
| | Phytosterols | Triterpene alcohols | Sesaminol | Tocopherols | Tocotrienols | Sesmolin† | Carotene‡ |
| Palmolein | 0.14 | 0.09 | – | 0.64 | 0.49 | – | 0.50 |
| Rice bran oil | 1.66 | 1.08 | – | 0.18 | 0.37 | – | – |
| Corn oil | 1.70 | 0.02 | – | 0.78 | – | – | – |
| Sesame oil | 0.56 | 0.17 | 0.85 | 0.07 | – | 0.4 | – |

†Hydrolysed to sesamol. ‡In red palm oil.

and riboflavin). Data on the phenolic antioxidant contents of Indian foods are rather limited and we have hardly any data on the flavonoid content of Indian foods. Indian foods rich in β-carotene and ascorbic acid are listed in Table 11. The foods rich in β-carotene also contain other carotenoids in greater or almost equal amounts. However, tocopherol is present mostly in cereals, nuts, oilseeds and edible oils (Table 12), and tocopherol intake is fairly satisfactory. Palm oil is the richest source, containing both tocopherols and tocotrienols. Similarly, rice bran oil is rich in tocotrienols, with the tocol content of other edible oils ranging from 400 to 800 mg (Table 12). The content of phenolic compounds in Indian foods (Table 13) and the polyphenolic content of diets (Table 14), determined as catechin equivalents, are fairly high. The antioxidant potential of these polyphenols have not yet been fully assessed, although their goitrogenic and iron absorption inhibition has been studied. Spices are another rich source of phenolic compounds and their antioxidant potential has been assessed at CFTRI and at the National Institute of Nutrition (NIN). We have, however, limited data on individual flavonoids, although they may have been estimated as phenolic compounds. Another group of foods rich in antioxidant chemicals is the non-glyceride component of edible oils, which contain plant steroids, tocopherols and tocotrienols and other antioxidants, such as sesamol and terpene alcohols (oryzanol) (Table 15). Red palm oil is a rich source of tocopherols and carotenoids (Table 16), while rice bran oil is rich in plant sterols that have hypocholesterolaemic properties. Rice bran oil also contains oryzanol, which can also act as an antioxidant and hypocholesterolaemic agent. Sesame oil contains the powerful phenolic antioxidant sesaminol (Table 15).

**Table 16.** Red palm oil as a source of carotene

| Carotene | Amount (μg/g) | β-Carotene equivalent |
|---|---|---|
| α-Carotene | 145 | 80 |
| β-Carotene | 310 | 310 |
| γ-Carotene | 20 | 10 |
| Lycopene | 10 | 0 |
| Xanthophyl | 15 | 0 |
| Total | 500 | 400 |

Spices, which are an invariable component of Indian diets, contain phenolic compounds that can act as powerful antioxidants. Eugenol from cloves, curcumin from turmeric and capsaicin from chillies have all been studied and shown to be effective antioxidants that can inhibit lipid peroxide-induced DNA damage.[22,23] The anticancer potential of turmeric and curcumin have been studied systematically by Kamala Krishnaswamy and her colleagues at NIN over the past decade.[24] They have also reported the antioxidant potentials of spices and their principles by *in vitro* methods and animal studies.

On the basis of available data on the antioxidant nutrient contents of foods and the dietary intakes of such foods, it is possible to assess the dietary antioxidant intake of different population groups in India. The daily intake of antioxidant nutrients by different socioeconomic groups are given in Table 17. The intake of nutrient antioxidants β-carotene, vitamin C and riboflavin does not even reach the RDA among the low socioeconomic groups in urban and rural India, let alone meet their antioxidant needs. In the well-to-do groups, however, intake of these nutrients is high enough to meet the RDA, but perhaps not high enough to provide adequate antioxidant protection. The daily intake of tocopherol, however, appear to be adequate across all groups, the average intake being 2–3 times its RDA. This means that the current dietary tocopherol intake of the Indian population can offer some protection against free radical damage. The intake of vitamin antioxidants is much lower, especially among preschool children of low income groups (Table 18). They suffer from various nutritional deficiencies more frequently than adults and hence are not well protected against oxidative stress. Their low intakes of fruits and vegetables are reflected in their low intakes of β-carotene and vitamin C. Consequently, their flavonoid antioxidant intakes may also be low. The intake of tocopherols derived from cereals, pulses and edible oils, however, appears to be satisfactory, as does the intake of phenolic antioxidants.[24] But how much protection these phenolic compounds offer as antioxidants is uncertain. It can be concluded that the poor-income population who consume unbalanced diets low in vegetables and fruits have dietary intakes of antioxidants that are too low to offer adequate protection against diseases caused by ROS. There is a need to determine the dietary antioxidant intake of the low-income groups of dietary antioxidants, apart from vitamin

**Table 17.** Daily antioxidant nutrient intake (per CU) by different socioeconomic groups in urban and rural areas of India

| Group | β-Carotene (μg) | Ascorbic acid (mg) | Riboflavin (mg) | Vitamin E (mg) |
|---|---|---|---|---|
| Urban | | | | |
| HIG† | 3524 | 98 | 1.5 | 29.9 |
| MIG | 2220 | 70 | 1.2 | 24.5 |
| LIG | 1328 | 47 | 0.9 | 18.3 |
| IL | 1408 | 40 | 0.9 | 18.5 |
| Slum | 992 | 40 | 0.8 | 13.2 |
| Rural (Monthly income, Rs.) | | | | |
| < 30 | 772 | 69 | 1.4 | 10.3 |
| 30–60 | 672 | 59 | 1.5 | 12.4 |
| 60–90 | 696 | 54 | 1.5 | 15.3 |
| 90–150 | 772 | 49 | 1.6 | 17.7 |
| >150 | 812 | 51 | 1.6 | 21.1 |
| Rural average | 736 | 55 | 1.5 | 13.5 |
| RDA | 2400 | 40 | 1.4 | 10.0 |

CU, consumption unit of an adult reference man; † HIG, high-income group; IL, industrial labour; LIG, low-income group; MIG, middle-income group; RDA, recommended dietary allowance.

**Table 18.** Daily dietary intake (per child) of antioxidants by preschool children in India

| Nutrient | Well-to-do group children | Low-income group children | RDA for preschool children |
|---|---|---|---|
| β Carotene (μg) | 3049 | 333 | 1600 |
| Ascorbic acid (mg) | 35.4 | 8.2 | 40 |
| Riboflavin (mg) | 1.72 | 0.5 | 0.9 |
| Vitamin E (mg) | 8.8 | 7.5 | 5.0 |
| Protein (g) | 39 | 25 | 26 |
| Methionine (g) | 0.88 | 0.52 | – |
| Cystine (g) | 0.55 | 0.39 | – |

RDA, recommended dietary allowance; –, no RDA.

**Table 19.** Detoxification of carcinogens by phytochemicals

| Phytochemical | Food source | Metabolic function |
|---|---|---|
| *Phase I metabolism* | | Metabolism, inducers of MFO, oxidation reduction, dehalogenation, desulphuration |
| Safrole | Cabbage | |
| Xanthones | Fruit | |
| Flavones | Vegetables | |
| Indoles | Tomatoes | |
| Coumaric acid | Strawberry | |
| Chlorogenic acid | Pineapple, green pepper | |
| *Phase II metabolism* | | Conjugation with glucuronic acid, glutathione, glycine and associated enzymes |
| Indole derivatives | Crucifera family | |
| Indole-3-carbinol | Cabbage, cauliflower | |
| Isothiocyanate | Broccoli | |
| Phenolic compounds | Black and green teas | |
| Flavanoles | | |
| Catechin | | |
| Theaflavin | | |
| 'Sulphorafone' (isolated) | Broccoli | |

antioxidants. The antioxidant intake of these low-income groups can be considerably augmented by including in their diets specific antioxidant-rich foods like red palm oil, rice bran oil and sesame oil.

**Detoxification of chemical toxicants and carcinogens by phytochemicals present in foods**

***Phytochemicals as detoxifying agents***

Another very important role of phytochemicals in foods in terms of disease prevention is their ability to detoxify drugs, toxins, carcinogens and mutagens. Phytochemicals detoxify toxicants in the body mainly by two mechanisms or phases;[25,26] phase I metabolism (or biotrans-formation) through reactions such as oxygenation, oxidation, reduction and dehydrogenation. Desulpheration in the microsomal mixed function oxidase (MFO) system introduces new functional groups into the hydrophobic toxic molecules, rendering them hydrophilic, and aids their rapid excretion. Several phytochemicals in foods act as harmless xenobiotics and induce enzymes for carrying out some of phase I metabolism. Phytochemicals and food sources that have been studied as promoters of phase I metabolism are given in Table 19. Paradoxically, the phase I drug-metabolising enzymes induced by these phytochemicals can also activate some biologically inactive compounds to become toxic derivatives (namely $CCl_4$), and they can also metabolise drugs rapidly and reduce their effectiveness in the treatment of diseases.

Phase II metabolism, which has a more important role in detoxification of xenobiotics (Table 19), involves conjugation reactions in which glucuronic acid, glutathione, glycine or amino acids are conjugated to the functional groups of toxic xenobiotics or their phase I metabolic products, rendering them even more polar and readily excretable. The enzymes involved in phase II metabolism are glutathione transferase, UDP glucoronyl transferase, NADPH quinone reductase and epoxide reductase.[27] These enzymes are induced by a variety of chemicals, including the phytochemicals present in food.

There have been extensive studies during the past two decades on the effect of phytochemicals present in food on drug-metabolising enzymes,[26–31] through which they offer protection against carcinogens and toxins. The balance between phase I and phase II enzymes is an important determinant of whether exposure to a carcinogen will result in toxicity or not.

The phytochemicals that are strong inducers of MFO are safrole, xanthenes, flavones and indoles. Rats maintained on a diet containing a higher content of indoles, such as a diet of cabbage sprouts, have a higher level of induced intestinal MFO activity and show an altered response to chemical carcinogens. Humans on similar diets show an enhanced hepatic metabolism of antipyrine. Coumarin derivatives present in fruits and vegetables (namely P-coumaric acid and chlorogenic acids present in tomatoes, strawberries, pineapple and green pepper) can induce glutathione-S-transferase and inhibit chemical carcinogenesis.

Vegetables of the crucifera family, such as cabbage, broccoli, brussel sprouts and cauliflower, contain a number of indole derivatives such as indole-3 acetic acid and isothiocyanates, which can act as anticarcinogens by inducing the phase II enzymes of drug metabolism. Recently, Talaly and coworkers from the National Institute of Health, Bethesda, Maryland, USA, have isolated an isothiocyanate named sulphorafane from broccoli, which is a powerful inducer of phase II drug-metabolising enzymes.[32,33] Screening of a wide range of vegetables[29] has indicated that broccoli and green onions have the highest activity with regard to induction of quinone reductase. Phenolic compounds in green and black tea (catechin, flavanols, thioflavin) have been reported to inhibit cytochrome P-450 MFO and enhance the phase II enzymes glutathione-S-transferase and quinone reductase.[34] These studies emphasise the importance of screening Indian vegetables, both familiar and unfamiliar, and Indian spices for their capacity to induce drug metabolising enzymes (DME), their anticarcinogenic potential and to isolate the the chemical concerned.

**Table 20.** Blocking and suppressing agents

| Agent | Phytochemical |
|---|---|
| Blocking | Glutathione; plant phenolic compounds; indoles; aromatic isothiocyanates; antioxidants. |
| Prevents carcinogens reaching the target tissue by sequestering them | |
| Suppressing agents | Isoprenoid compounds in fruits, vegetables, cereal grains and essential oils; allelic sulphides of garlic; 'agoene', prevents platelet aggregation, protease inhibitors, plant sterols and caffeine. |
| Inhibit carcinogenesis, presumably acting at the cellular level, when administered subsequent to or just prior to exposure to a carcinogen | |
| Suppress tumour growth by inhibiting HMG-CoA reductase and cholesterol biosynthesis | |
| Induce Phase II drug-metabolising enzymes | |
| Protect against cardiovascular disease by inhibiting cholesterol biosynthesis | |

HMG-CoA, β-hydroxy β-methyl glutaryl coenzyme A.

**Table 21.** Inhibitor compounds of carcinogenesis occurring in plant foods

| Category | Inhibitory compounds |
|---|---|
| Compounds preventing formation of carcinogens from precursor compounds | Ascorbic acid, α- and r-tocopherols, caffeic acid, ferrulic acid<br>Phenols (e.g., ellagic acid, caffeic acid, ferrulic acid, P-hydroxy cinamic acid)<br>Indoles (e.g., indole-3-acetonitrile, indole-3-carbinol, 3-3′ indolymethane)<br>Aromatic isothiocyanates (e.g., benzyl isothiocyanates, phenthyl isothiocyanates)<br>Coumarins (e.g., courmarin, limethin)<br>Flavones (e.g., quercetin pentamethyl ether)<br>Diterpenes (e.g., kahweel palmitate)<br>Retinoids and carotenoids (e.g., retinyl palmitate, retinyl acetate, β-carotene |
| Suppressing agents | Soybean protease inhibitor, benzyl-isothiocyanate, B-sitosterol, caffeine, fumeric acid, selenium |

**Table 22.** Neuropharmacological agents

| Agent | Food source |
|---|---|
| 5-Hydroxytryptamine | banana |
| Tyramine | annane |
| Dopamine | tomato |
| L-Dopa | avocado |
| Opiod compounds | spices (e.g., pepper) |
| Noradrenaline | herbs (e.g., ephydra) |

Some work on DME in spices has been carried out at CFTRI, NIN and other laboratories in India. Such work must be pursued with greater intensity.

**Blocking and suppressing agents in cancer prevention**

According to Wattenberg,[30,31] several minor chemical constituents of foods inhibit carcinogenesis by acting as blocking and suppressing agents. Besides enhancing metabolic disposal of carcinogens as discussed above, the blocking agents also prevent active carcinogens reaching the target tissue or cell by sequestering them (e.g., glutathione, plant phenolic compounds). The suppressing agents are those that inhibit carcinogenesis, presumably by acting at the cellular level, when administered subsequent to or prior to exposure to a carcinogen, and in the absence of which cancer would result. Compounds that act as blocking or suppressing agents are given in Table 20.

Besides inducing phase II drug-metabolising enzymes, isoprenoid compounds in fruits, vegetables, cereal grains and essential oils (namely limonene) also act as suppressing agents. They suppress tumour growth by inhibiting β-hydroxy β-methyl glutaryl coenzyme A (HMG-CoA) reductase activity, a rate limiting step in cholesterol biosynthesis.[35] Such inhibition depletes cells of intermediate products required for cell proliferation. By inhibiting cholesterol synthases these compounds can also protect against cardiovascular disease. Garlic (*Allium sativum*), widely used in India as a spice, contains sulphur compounds. Allelic sulphide can induce phase II drug-metabolising enzymes to inactivate carcinogens. After a mild processing, garlic has been shown to yield a compound called agoene that can prevent platelet aggregation.[36] This action of garlic may be related to the reported beneficial effect of garlic in heart disease.

Several compounds that have antioxidant properties (ascorbic acid, tocopherols, phenols) can also inhibit the formation of carcinogens from their precursors. Indoles and aromatic isothiocyantes are important inducers of DME. Phenols can also bind carcinogens to prevent their action at the cellular level. Protease inhibitors, plant sterols and caffeine can act as suppressing agents. Although these compounds occur widely in foods, as shown in Table 21, the content of several of these compounds in a number of Indian foods is yet to be determined.

The contents of total phenolic compounds and thiocyanates have been determined in Indian foods (Table 13). It must be remembered that both of these class of compounds bind dietary iron, making it unavailable. Thiocyanate compounds, as well as phenols, are goitrogens. Thiocyanates, isothiocyanates and indoles are powerful inducers of DME. The former two occur in vegetables belonging to the crucifera family, namely cabbage and mustard/rape greens and seeds. These chemicals present in foods can be separated by HPLC and gas chromatography (GC). Their inhibitory potential can be tested both *in vitro* and *in vivo* on enzyme systems that actively convert procarcinogens to carcinogens, by their effects on DME, and by their role in sequestering carcinogens and preventing their binding to cell membranes and DNA.

**Neuropharmacological chemicals in foods**

A number of biogenic amines, such as 5-hydroxy tryptamine, noradrenaline, tyramine, dopamine and their precursors, are reported to be present in small quantities in foods such as bananas, annanas, tomatoes and avocados, and in spices such as pepper (Table 22). The pharmacological potential and health significance of these biogenic amines naturally present in foods is not yet clear. A notion widely prevalent in ancient systems of medicine (e.g., charaka samhita) was that some foods have special effects on brain function, learning and behaviour, and that they sharpen mental agility. It is possible

that some of these neuroactive chemicals can contribute to the dietary effect on mental and brain function. This is another area where the effect of specific foods on mental function and the phytochemicals responsible for such action requires in-depth study. Fabia beans being rich in L-dopa are used in the treatment of Parkinson's disease, which is caused by a deficiency of dopamine.[37] Studies on malnourished children have shown that anaemia, PEM and vitamin A deficiencies are also associated with impaired brain and mental function and learning ability. It is possible that the observed impairment of mental function in such children may be due to an imbalanced diet deficient in many neuroactive phytochemicals.

**Plant sterols**

Phytosterols such as stigmasterol, sitosterol and campesterol are widely distributed in the plant kingdom and in plant foods. These plant sterols get concentrated in the non-glyceride portion of edible oils and have been shown to have hypocholesterolaemic potential. Rice bran oil is a good example of a food with a high sterol content and hypolipidaemic activity (Table 15). Rice bran oil has a high level of an unsap fraction (3–4%) and has been shown to possess a hypocholestrolaemic effect both in experimental animals[38] and in patients with hypocholesterolaemia. The cycloarterol present in the non-glycemide component of rice bran oil also has a cholesterol-lowering property. Some unconventional oils have an unsap component of up to 10% and may have powerful blood cholesterol lowering potency, an aspect that requires further study.

**Phytochemicals and immune function**

It is believed that some phytochemicals have the potency for augmenting both humoral and cell-mediated immune function. This aspect of functional foods and their components has not yet been studied fully. Recently, it has become apparent that both vitamin C and carotenoids may have beneficial effects on immune function, thereby reducing cancer risk by enhancing the tumour surveillance of the immune system.[39–41]

**Other phytochemicals**

Besides the chemicals with specific functions present in plant food, plant foods also contain a wide range of other chemical compounds, such as acids, esters, bases and phenolic compounds, that impart taste and flavour to food. Most foods, particularly fruits, vegetables and spices, contain very small quantities of a wide range of volatile compounds, such as alcohols, esters, ketones, aldehydes and terpenes, that are responsible for the flavour and aroma of foods. It is not yet clear whether these compounds also have any beneficial biological function in the body. They may have a role in stimulating appetite and satiety. Terpenes have, however, been shown to act as anticancer agents.[35]

**Current research on bioactive phytochemicals**

Research interest in phytonutrients and their role in disease prevention is currently being pursued with great vigour. New interactions are being recognised between plant foods and the phytonutrients present in them. For example, Japanese women have rates of breast cancer that are lower than women from other parts of the world. One of the dietary factors recognised as being responsible for this difference is the presence of high levels of soy products in the typical Japanese diet. Soy beans contain high levels of phytoestrogens, oestrogen-like compounds that are believed to block the action of human oestrogen in the body, lowering the risk of breast cancer.[3]

**Desirable dietary intake of phytonutrients**

Plant foods contain a large number of phytonutrients that have the potential for offering protection against a range of non-communicable diseases like diabetes, cancer, cardiovascular disease and cataract. Some vitamins in foods also have similar protective functions. As in the case of nutrients, there is a need to determine the desirable dietary intake of these phytochemicals to provide maximal protection against the above-mentioned diseases. As shown in the case of some nutrients (Table 9), the intake of these nutrients have to be several-fold higher than the RDA to offer protection against these diseases. As a prelude to arriving at desirable dietary intakes of phytonutrients, there is a need for precise quantitative data on the content of these phytochemicals in commonly consumed foods and their bioavailability and biological potency. Such a knowledge will also be useful in formulating special diets towards preventing a particular disease entity. As discussed above, several phytochemicals in foods have similar biological effects (e.g., antioxidant properties) and the same compound may elicit different biological responses (e.g., antioxidant and DME induction). The sum total effect of these compounds in a diet composed of different foods can be additive and may often be synergistic. These aspects of dietary content of phytochemicals have to be taken into account when recommending their desirable dietary intake.

There is a need to set up an *in vivo* system to test the biological potency of a given diet with a particular biological property (antioxidant protection or induction of drug metabolising enzymes, inhibition of carcinogenesis). Future research in the science of nutrition may be profitably directed towards evaluating the potency of phytochemicals in foods and diets and their health implications.

**References**

1. Indian Council of Medical Research. Medicinal Plants of India, Vol. I and II. New Delhi: Indian Council of Medical Research, 1987.
2. Beagley S. Beyond vitamins. News Week, USA. April 24, 1994; 43.
3. Anon: The function of foods. Food Facts Asia 1999; 6: 1–3.
4. Anon: Round the World: USA: Bionutrition NIH strategies plan. Lancet 1993; 341: 1336.

5. Tharanath RN, Muralikrishna G, Salinath PV, Rao R. Plant carbohydrates: overview. Proc Natl Acad Sci (Plant Sci) 1987; 97: 81–155.
6. Birch GG, Parker KJ, eds. Dietary Fibre. London: Applied Science Publications, 1982.
7. Schnecman BO. Dietary fibre: scientific status summary. Food Tech 1989; 43: 133–139.
8. Narasinga Rao BS. Dietary fibre in Indian diets and its nutritional significance. Bull. Nutr. Foundation of India 1988; 9: 1–5.
9. Annual Report 1992-93, National Institute of Nutrition, Hyderabad, India.
10. Sharma RD, Raghuram TC. Hypoglycemic effect of fenugreek seeds in non-insulin dependent diabetic subjects. Nutr Res 1990; 10: 731–739.
11. Packer L, Glazer AN, eds. Oxygen radicals in biological systems, Part B. Methods in Enzymology, Vol. 186. New York: Academic Press, 1990.
12. Halliwell B. Antioxidants in human health and disease. Ann Rev Nutr 1996; 16: 33–50.
13. Strain JJ, Benjic IIF. Diet and antioxidant defence. In: Sedler MO, ed. Encyclopedia of Human Nutrition, Vol. 1. New York: Academic Press, 1999; 95–105.
14. Anon: Role of antioxidants in cancer prevention and treatment. Vitamin E Research Summary 1991 (Sept): 1–15.
15. Krinsky NI. Action of carotenoids in biological systems. Ann Rev Nutr 1993; 13: 561.
16. Packer L, ed. Carotenoids, Part A. Chemistry, separation, quantitation and antioxidation. Methods in Enzymology, Vol. 213. New York: Academic Press, 1964.
17. Harborne JB, ed. Biochemistry of Phenolic Compounds. New York: Academic Press, 1964.
18. Kuhnau. The flavonoids: a class of semiessential food components, their role in human nutrition. World Rev Nutr Diet 1976; 24: 117–191.
19. Ziegler RG, Subar AF, Craft NE, Ursin G, Patterson BH, Graubard BI. Does β-carotene explain why reduced cancer risk is associated with vegetable and fruit intake. Cancer Res. 1992; 52: 2060S–2066S.
20. Hertog MG, Feskens EJM, Hollman PCH, Katan MB, Kromhout D. Dietary anitoxidant flavanoids and the risk of coronary heart disease: Zulfan study. Lancet 1993; 342: 1007–1011.
21. Frankel EN, Kenner J, German JB, Parks E, Kinsella JE. Inhibition of oxidation of human low density lipoprotein by phenolic substances in red wine. Lancet 1993; 341: 453–457.
22. Shalini VK, Srinivas L. Lipid peroxide induced DNA damage: Protection by turmeric (*Curcuma longa*) Mol Cell Biochem 1987; 77: 3–10.
23. Reddy AC, Lokesh BR. Studies on spice principle as antioxidants in the inhibition of lipid peroxidation of liver microsomes. Mol Cell Biochem 1992; 111: 117–124.
24. Krishnaswami K, Raghuramulu N. Bioactive phytochemicals with emphasis on dietary practices. Ind J Med Res 1998; 108: 167–181.
25. Narasinga Rao BS, Prabhavati T. Tannin content of foods commonly consumed in India and its influence on ionizable iron. J Sci Food Agric 1982; 33: 89–96.
26. Parke DV, Ionnides C. The role of nutrition in toxicology. Ann Rev Nutr 1981; 1: 207–234.
27. Guengrich FP. Diet, nutrition and metabolic regulation: effect of nutrition factors involving bioactivation and detoxification of chemicals. Ann Rev Nutr 1984; 4: 207–231.
28. Talalay P. Mechanisms of induction of enzymes that protect against chemical carcinogenesis. Adv Enzyme Reg 1980; 28: 237–250.
29. Prochaska H, Santamaria AB, Talalay P. Rapid detection of inducers of enzymes that protect against carcinogens. Proc Natl Acad Sci USA 1992; 89: 2394–2398.
30. Wattenberg LW. Inhibition of carcinogenesis by minor nutrient constitutents of the diet. Proc Nutr Soc 1990; 49: 173–183.
31. Wattenberg LW. Inhibition of carcinogenesis by minor dietary constitutents. Cancer Res 1992; 52: 2085S–2091S.
32. Zhang Y, Talalay P, Cho CG, Posner GH. A major inducer of anticarcinogenic protective enzymes from broccoli: isolation and elucidation of structure. Proc Natl Acad Sci USA 1992; 89: 2399–2403.
33. Zhang Y, Kensler TW, Cho CG, Poner GH, Telalay P. Anticarcinogenic activities of sulforaphane and structurally related synthetic norboronyl isothiocyanates. Proc Natl Acad Sci USA 1994; 91: 3147–3150.
34. Boone CW, Wattenberg LW. Current strategies of cancer chemoprevention: Report of proceedings of a seminar. Cancer Res 1994; 54: 3315–3318.
35. Elson CE, Yu SG. The chemoprevention of cancer by mevalonate-derived constitutents of fruits and vegetables. J Nutr 1994; 124: 607–661.
36. Jain MK, Aptiz-Castro R. Garlic – A product of spilled ambrosia. Curr Sci 1993; 65: 148–156.
37. Kempster IPA, Walquist ML. Dietary factors in the management of Parkinson's disease. Nutr Rev 1994; 52: 51–59.
38. Sharma RD, Rukmini C. Hypocholesterolemic activity of unsaponifiable matter of rice-bran oil. Ind J Med Res 1989; 85: 278–281.
39. Bendit A. Carotenoids and immune response. J Nutr 1989; 119: 112–115.
40. Henson DE, Block G, Levin M. Ascorbic acid biological function in relation to cancer. J Natl Cancer Inst 1991; 83: 547–550.
41. Ringer TV, Deloof MJ, Winterrowd GE, Francom SF, Gaylor SK, Ryan JA, Sanders ME, Huges GS. β-carotene: Effect on serum β-lipoproteins and immunological indices in humans. Am J Clin Nutr 1991; 53: 688–694.