# New Food Ingredients and Pharmaceutical Foods

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## Abstract

Food can be designed to possess unique functional properties to meet the nutritional needs of a wide range of consumers. Original examples of such functional or pharmaceutical foods include Vitamin D fortified milk, enriched breads and iodized salt. Today, ingredients are commonly used to alter the nutritional composition of food products to decrease or replace fats, sugar and sodium or to supplement foods with minerals, fiber and unsaturated fatty acids. More recently, numerous biologically active components, endogenous to foods, have been proposed to be used as food ingredients. These compounds include antioxidants such as phenolics, dipeptides and carotenoids and anticarcinogens such as conjugated linoleic acid and thiosulfinates. Aspects of the practicality of designing foods/diets high in these biologically active compounds including labeling/regulatory restrictions, incorporation and stability in food products and interactions with other dietary components will be discussed.

## Introduction

Pharmaceutical foods, hyperfoods, designer foods, functional foods and nutraceuticals are all terms designed to describe the concept of altering raw food composition or formulating processed foods to provide maximal nutritional benefits to consumers. While the use of these terms is relatively recent, the concept of functional foods has actually been a reality in the food industry for decades as products such as flour, bread, breakfast cereal and even salt have been fortified with essential vitamins and minerals. The Food and Drug Administration currently considers fortification appropriate to prevent widespread nutrient deficiency (e.g. iodine in salt); restore nutrients lost in processing (e.g. enriched flour and rice); balance the total nutritional profile with energy content (e.g. breakfast cereals); and to assure that substitute foods are nutritionally similar to the foods they replace (e.g. Vitamin A supplementation of margarine). Food such as candies, carbonated beverages and snack foods are not deemed as appropriate for fortification by the FDA.

The concept of functional or pharmaceutical foods describes foods which go beyond the practice of fortification to meet daily nutritional requirements. Functional foods would contain either large amounts of vitamins or minerals or elevated levels of nonessential dietary components which are believed to possess health benefits. Functional foods are already widespread and popular in Japan where foods and beverages containing compounds such as ginkgo leaves, oyster extract, oligosaccharides, peptides and ginseng. Functional foods supplemented with compounds such as fiber, polyunsaturated fatty acids, isotonic mixtures of sugars and minerals and live yogurt cultures are common in the U.S.. Even foods designed to be lower in fat, sodium and cholesterol could be considered functional foods. Unfortunately, the origin of many functional foods in the U.S. market is the direct result of the newest health fads and often the nutritional benefits of these foods is unknown.

Current interest in functional foods has concentrated on food components which have the potential to inhibit the development of common diseases. One such example is calcium supplementation of breads and orange juice to provide nutritional defense against osteoporosis. While vitamin and essential mineral supplementation has a long history, recent interest in functional foods has concentrated on many nonessential food components which have been shown to decrease the risk of cancer, heart disease and intestinal disorders. The purpose of this review will be to explore several classes food components which possess biological activity. Problems associated with the production of functional foods in relation to food processing, nutritional, biochemical and regulatory issues will also be discussed.

## Phenolics

Phenolic compounds in foods include simple phenols and phenolic acids, hydroxycinnamic acid derivatives and the flavonoids (Ho, 1992). These phenolic classes contain numerous compounds which are widely distributed in plant foods (Table 1). Since phenolics are found in virtually all plant foods often at very high concentrations, these compounds are consumed in quantities of up to a gram per day (Huang and Ferraro, 1992). Phenolic compounds influence the quality, acceptability and stability of foods by acting as flavorants, colorants and antioxidants. The presence of conjugated ring structures and hydroxyl groups allows phenolics (Figure 1) to act as antioxidants (Graf, 1992; Shahidi et al., 1992). In addition to the ability of phenolic compounds to inhibit lipid oxidation in biological systems, pure phenolic compounds and crude extracts from rosemary and green tea can inhibit oxidative reactions in processed foods (Shahidi et al., 1992). However, flavors associated with these extracts often limits their utilization as food additives. Phenolic compounds exhibit anticarcinogenic activity in numerous animal models (for review see Huang and Ferraro, 1992). The antioxidant activity of phenolics is thought to be involved in their anticarcinogenic properties. However, phenolic compounds could also be anticarcinogenic due to their ability to reduce the bioavailability of carcinogens, inhibit the metabolic activation of carcinogens, inhibit arachidonic acid metabolism, and inhibit of protein kinase C activity (Huang Ferraro, 1992).

The anticarcinogenic activity of phenolics has been correlated with inhibition of colon, esophagus, lung, liver, mammary, and skin cancer. Several examples of phenolic compounds which inhibit carcinogenesis include curcumin, quercetin, coumarins, caffeic acid, ellagic acid and catechin derivatives (Ho, 1992). The catechin derivatives, epigallocatechin, epigallocatechin gallate, epicatechin, epicatechin-3-gallate and gallocatechin are found in concentrations as high as 10.0% of the dry weight of green tea leaves (Balentine, 1992). In addition to catechins, green tea contains gallic acid, coumaric acid, caffeic acid and quercetin which also have antioxidant and anticarcinogenic properties. The positive antioxidant and anticarcinogenic profile of the molecular components of green tea has prompted extensive research on the health benefits of green tea extracts. Topical or oral administration of green tea extracts has been reported to inhibit mouse skin (Katiyar et al., 1993) and mouse forestomach and lung (Wang et al., 1992) cancer. While the majority of research indicates that green tea extracts exhibit anticarcinogenic activity, Hirose and coworkers (1992) found that green tea extracts can simultaneously inhibit small intestine and promote liver cancer in a rat multi-organ carcinogenesis model. This research along with reports of the carcinogenic activity of caffeic acid in rat forestomach epithelium and kidney, sesamol in rat forestomach, and catechol in rat stomach (Ito et al., 1992) suggest that

substantially increasing the consumption of phenolic compounds may not be prudent until the anti- and pro-carcinogenic activities are better understood.

Phenolic compounds have also been associated with the inhibition of atherosclerosis. The association of dietary phenolics (especially in the form of red wine) and coronary heart disease (CHD) was observed in segments of the French population who have similar atherogenic risk factors (saturated fat intake and plasma cholesterol levels) as segments of the U.S. population but have much lower incidence of CHD (Renaud and de Lorgeril, 1992). The positive effect of red wine on CHD could be due to the effects of ethanol and/or the ability of phenolic compounds to inhibit lipid oxidation (Kinsella et al., 1993).

Oxidation of low density lipoprotein (LDL) is thought to result in increased accumulation of LDL into foam cells resulting in the acceleration of atherosclerotic plaque formation (Steinberg et al., 1989). Antioxidant nutrients (vitamins C and E, ß-carotene and selenium) have been postulated to decrease the oxidation of LDL and thus decrease the risk of atherosclerosis (for review see Esterbauer et al., 1992). Phenolic compounds from red wine inhibit the oxidation of LDL more effectively than a-tocopherol suggesting that red wine could decrease oxidation-induced atherosclerosis (Frankel et al., 1993; Kanner et al., 1994). Phenolic compounds in red wine which inhibit oxidative reactions include catechins, flavonols, anthocyanins and tannins. Since these phenolics are also found in a large number of other fruits and vegetables, these nonessential antioxidants could be partially responsible for the positive effects of fruit and vegetable consumption on CHD. Unfortunately, limited absorption data is available on dietary phenolic compounds.

## Conjugated Linoleic Acid

Conjugated linoleic acid (CLA) is a term given to a group of linoleic acid (18:2 n-6) isomers in which the double bonds are conjugated instead of in the typical methylene interrupted configuration. Nine different positional and geometric isomers of CLA have been reported as minor components of a variety of food products (Ha et al., 1989). Interestingly, food lipids originating from ruminant animals (beef, dairy and lamb) contain high levels of CLA of which the 9-cis, 11-trans isomer predominates. CLA concentrations in dairy products range from 2.9 - 11.3 mg/g fat of which the 9-cis, 11-trans isomer makes up 73 - 93% of the total CLA while beef fat contains 3.1 - 8.5 mg/g fat with the 9-cis, 11-trans isomer contributing to 57 - 85% of the total CLA (Chin et al., 1992; Shantha and Decker, 1993; Shantha et al., 1993). CLA concentrations in fats from nonruminants and vegetable oils typically ranges from 0.6-0.9 mg/g fat; Chin et al., 1992). The high proportion of 9-cis, 11-trans CLA in fats from ruminants is believed to be due to a specific geometric and positional bioisomerization of linoleic acid by ruminal bacteria (Kepler et al., 1966).

CLA has been shown to inhibit the development of mouse epidermal (Ha et al., 1987), mouse forestomach (Ha et al., 1990), and rat mammary cancer (Ip et al., 1991; Ip et al., 1994). The anticarcinogenic mechanism of CLA is not well understood, however, CLA has been found to be an effective antioxidant *in vitro* (Ha et al., 1990). Further research is needed to elucidate the antioxidant mechanism of CLA and to determine if CLA's antioxidant activity plays a role in its ability to inhibit cancer and atherosclerosis.

Interestingly, feeding mice (Ha et al., 1990) and rats (Ip et al., 1991) a mixture of CLA isomers results in the preferential incorporation of the 9-cis, 11-trans CLA into membrane phospholipids

suggesting that 9-cis, 11-trans CLA is the biologically active isomer. Extrapolation of dietary CLA concentrations which are effective in animal models indicates that equivalent CLA concentrations in a 70 kg human would be in the order of 1.5 - 3.0 g of CLA per day which is significantly higher than the estimated consumption of approximately several hundred mg/person/day in the U.S. (Ip et al., 1991). It is possible that CLA concentrations could be increased by manipulation of the nutritional regimes of livestock or by food processing technology. However, foods high in CLA are also high in fat therefore one must weigh the benefits of dietary CLA with the deleterious effects of fat consumption.

## Histidine-Containing Dipeptides

Carnosine (ß-alanyl-L-histidine) and anserine (ß-alanyl-L-1-methyl-histidine) (Fig. 2) are dipeptides found in skeletal muscle. The predominance of carnosine and anserine in skeletal muscle is species dependent (Crush, 1970; Table 2). For example, there is essentially no anserine in human muscle. In pigs, beef, goat and turkey, carnosine concentrations are higher than anserine. However, in salmon, rabbit and chicken skeletal muscle, anserine is predominant. Boldyrev and his co-workers (1987) were the first to report that carnosine and anserine inhibit the oxidation of lipid membranes. Since then, other researchers found that carnosine was capable of inhibiting lipid oxidation catalyzed by a variety of oxidation catalysts in numerous lipid systems with significant antioxidant activity occurring at concentrations comparable with those in skeletal muscle tissues (for review see Chan and Decker, 1994).

Carnosine is absorbed intact in the jejunum of the small intestine by a specific active transport system in brush border membranes (Ferraris et al., 1988). Carnosine and anserine are dispersed into the blood where they were transported to kidney, liver and skeletal muscle (Abe, 1991). Absorbed carnosine is either utilized by peripheral tissue or is hydrolyzed into ß-Ala and His by carnosinase, which is present in the blood, kidney and liver (Wolos et al., 1982). The kidney seems to be the main organ responsible for the catabolism and excretion of the dipeptides. Even though 0.05 - 0.25 g of carnosine are consumed daily (based on diet containing 100 g pork, beef or chicken/day) very little data is available on the dynamics of carnosine absorption, transport and catabolism.

Endogenous skeletal muscle carnosine concentrations can be affected by diet. Dietary histidine deficiency in rats reduced skeletal muscle carnosine concentrations (Tamaki et al., 1984). Increases in skeletal muscle carnosine concentrations (2.8 fold) have been observed in rats supplemented with 5% histidine (Tamaki et al., 1984). Dietary supplementation of carnosine also influences skeletal muscle carnosine concentrations. Low dietary carnosine supplementation (0.9%) did not significantly increase carnosine concentrations in skeletal muscle of rat while elevated dietary carnosine concentrations (5%) doubled skeletal muscle carnosine concentrations (Tamaki et al., 1984). Even though carnosine is incapable of regenerating the a-tocopherol radical (Gobunov and Erin, 1991) an indirect relationship between carnosine and a-tocopherol seems to exist *in vivo* as evidenced by a-tocopherol deficiency resulting in decreased rabbit skeletal muscle carnosine concentrations (McManus, 1960).

Carnosine (2 mM) is capable of inhibiting copper-catalyzed oxidation of LDL suggesting that it could inhibit the development of atherosclerosis (Bogardus et al., 1993). In addition, dietary carnosine (0.825%, calculated to be equivalent to a 25% beef diet) is capable of inhibiting 7,12-dimethylbenz[a]anthracene-induced breast cancer in vitamin E deficient rats (Boissonneault et

al., 1994; Fig 3.). Dietary carnosine extended the time to 50% incidence of palpable tumors from 12.7 weeks for the control (no supplemented carnosine or a-tocopherol acetate) to 18.9 weeks. The time to 50% tumor bearing animals with carnosine supplemented treatment was also greater than a-tocopherol acetate (50 ppm) or a-tocopherol acetate + carnosine supplemented animals. Whether the antioxidant properties of carnosine is involved in its anticarcinogenic properties has yet to be determined.

## Garlic

Both fresh and processed garlic (*Allium sativum*) have been used as food flavorants and medicinal aids as long ago as 1500 BC. Louis Pasteur and Albert Schweitzer described antibacterial and antiparasitic properties of garlic in the 1800's and more recently garlic juice has been found to be potent antimicrobial agents inhibiting the growth of bacteria, fungi and yeasts (for review see Block, 1985). In addition to antimicrobial properties, garlic is thought to have positive influence against cardiovascular disease by inhibiting blood clotting and lowering blood cholesterol and lipid concentrations (Lin, 1992). Garlic has also been found to possess anticarcinogenic activity inhibiting the formation of colon, skin, esophageal, liver and forestomach cancers (Wargovich, 1994).

The medicinal properties of garlic is closely related to its flavor components. Studies over the past 150 years has revealed that the fresh odor of garlic produced upon cutting or crushing is the result of the sulfur containing compound, (+)-S-allyl-L-cysteine sulfoxide or alliin being enzymically converted by allinase to an unstable compound known as allicin. Allicin can degrade into various mono-, di-, and trisulfide thioesters or three molecules can condense into a compound known as ajoene (Block, 1985). These strong smelling volatile compounds, several nonvolatile components (mostly derivatives of cysteine) as well as whole, extracted and deodorized garlic products have been reported to inhibit cancer (Wargovich, 1992) and cardiovascular disease (Lin, 1994). The anticarcinogenic activity of garlic has been attributed to the allicin breakdown products which contain allyl side chains. The anticarcinogenic activity of diallyl sulfide, the most active of these compounds, is thought to involve its ability to inhibit bioactivation of carcinogens. The ability of garlic to inhibit cardiovascular disease involves several potential mechanisms. Garlic derivatives have been found to lower blood cholesterol and lipid levels in chickens and rats presumably by blocking hepatic cholesterol and fatty acid synthesis (Qureshi, 1990; Lin, 1994).

Garlic also has the ability to inhibit blood clotting and cause vasodilation (Lin, 1987; Block, 1985). While preliminary research on the biological activity of dietary garlic is promising, little information is available on the ability of humans to absorb dietary sources of these phytochemicals. Therefore, before dietary recommendations can be made, the nutritional significance of garlic must be more thoroughly investigated.

## Lactic Acid Bacteria

Consumption of live lactic acid cultures has long been promoted as beneficial to health. Lactic acid cultures are traditionally used in fermented dairy and meat products including yogurt, cheeses, buttermilk and fermented sausage. Use of lactic acid fermentations originally developed

from the desire to improve the flavor, texture and shelf-life of foods, however, live lactic acid cultures are also added to nonfermented foods for their potential health benefits. Such an example of a nonfermented food containing live lactic acid bacteria cultures is acidophilus milk. The ability of dairy products to buffer stomach pH is believed to allow passage of live organisms into the lower gastrointestinal (GI) tract. Recently, addition of lactic acid cultures to other foods such as orange juice has been proposed (O'Donnell, 1994), however, it is unknown whether the bacteria will survive the conditions present in these foods or whether these types of products will allow passage of live organisms into the lower GI. Unfortunately, substantial research evidence on the health benefits of lactic acid bacteria is often lacking for many of the health claims currently being promoted.

Consumption of live lactic acid bacteria has been suggested to positively influence health problems such as lactose intolerance, diarrhea, hypercholesteremia, cancer, immune system stimulation, constipation and vaginitis (Sanders, 1994). Of these proposed benefits, only alleviation of lactose intolerance by lactic acid bacteria is supported by strong experimental evidence in human subjects. Fermented dairy products containing live lactic acid cultures and high lactose loads can often be consumed by lactose intolerant individuals without development of adverse symptoms. Lactase associated with the bacteria is thought to aid the digestion of lactose in the intestine. Sonication of the lactic acid bacteria can increase the rate of lactose digestion in the GI presumably by increasing the ability of lactase to reach its substrate. The ability of yogurt to aid the digestion of lactose is diminished by heat treatments. It is unclear whether this is due to reduced cell viability or heat inactivation of lactase (McDonough et al., 1987). Live dairy cultures are not capable of aiding the digestion of lactose in excess to that normally present in dairy products.

Lactic acid cultures have also been suggested to have a positive influence on immune system response presumably by stimulating gut associated lymphatic tissue. Consumption of live lactic acid cultures could provide an important immuno stimulant for individuals exposed to extensive or prolonged antibiotic treatments. Anticarcinogenic activity has also been related to lactic acid bacteria. Bacterial populations in the colon can cause the formation of carcinogenic or mutagenic compounds such as nitrosoamines, phenols, cresols, idole, aglycones and secondary bile acids (Tomomatsu, 1994). These compounds are produced by bacteria including *Escherichia coli, Streptococcus faecalis*, and clostridia which contain high activity of the enzymes azoreductase, nitroreductase and ß-glucuronidase. Reducing the population of these potentially harmful bacteria in the GI is thought to decrease the risk of colon cancer. Lactic acid bacteria have been found to decrease the activity of azoreductase, nitroreductase and ß-glucuronidase in feces. The mechanism of reduced enzyme activity by lactic acid bacteria is not clear but has been postulated to be due to a lowering of bowel pH by bacterial acid production or by out competing the putrefactive bacteria (Sanders, 1994).

## Oligosaccharides

Several oligosaccharides have been reported to possess unique physiological properties. These bioactive carbohydrates are generally short chained polysaccharides which are not digestible by human enzymes but are consumed by intestinal bacteria such as bifidobacteria and lactic acid bacteria. Most physiologically relevant oligosaccharides contain fructose, glucose and galactose which are linked through nondigestible ß 1-4, a 1-3 and ß 1-2 bonds (Oku, 1994; Tomomatsu,

1994). These polysaccharides can either be natural (e.g. raffinose and stachyose), synthetically produced from monosaccharides or derived from the hydrolysis of natural polysaccharide sources such as soybeans. Most oligosaccharides exhibit good solubility in water, low viscosity, and low to moderate sweetness.

Consumption of oligosaccharides results in an increase in beneficial bacterial populations and a reduction in the growth of putrefactive bacteria in the lower GI thereby decreasing the production of toxic and carcinogenic compounds associated with these bacteria. Selection of a more beneficial intestinal bacterial population by oligosaccharides has been suggested to decrease the risk of colon cancer, decrease pathogenic diarrhea, and protect liver function by decreasing formation of toxins such as ammonia in the GI (Tomomatsu, 1994). Oligosaccharides have also been suggested to be used as a sugar substitute which does not affect insulin secretion (monosaccharides are not produced) and does not promote dental caries (Oku, 1994).

Use of oligosaccharides in foods is easier than use of live bacteria cultures which often can not survive processing operations, storage and/or the acid conditions of the stomach. Oligosaccharides are also easier to use in foods than dietary fiber since they are water soluble and do not influence the viscosity or water binding properties of foods. An additional advantage of oligosaccharides is that they exhibit beneficial effects at concentrations as low as 3 g/day (Oku, 1994). However, oligosaccharides can cause diarrhea at high concentrations and can produce flatulence under certain circumstances. Several products containing oligosaccharides are currently available and are recognized by the FDA as "generally recognized as safe" food ingredients. These food additives have been extremely popular in Japan but have yet to gain widespread acceptance in North America.

## Challenges Facing the Future of Functional Foods

The production of functional, pharmaceutical or designer foods which have the potential to prevent or treat diseases has great consumer appeal. This interest will only increase with increases in experimental data on the physiological benefits of food components and with increases in the average age of the U.S. population and health care costs. While functional foods are a reality in countries such as Japan, several major problems and hurdles exist before these products can become a reality in the U.S. food market.

The major hurdle facing the future of functional foods is current labeling regulations. The Nutrition Labeling and Education Act of 1990 allows food manufacturers to relate the consumption of selected nutrients to different health claims. These claims include: calcium and osteoporosis; fat and cancer; saturated fat and cholesterol and coronary heart disease; fruit, vegetable and grain products and cancer; fruit, vegetable and grain products (soluble fiber) and coronary heart disease; and sodium and hypertension (Mermelstein, 1993). While food manufacturers are allowed to make statements about the relationship between these nutrients and diseases, no degree of risk reduction can be stated and only "might" or "may" can be used when discussing the relationship (e.g. diets low in sodium may reduce the risk of hypertension).

Another problem with the production of functional foods is the ability of food companies to patent natural food components. Without the guarantee that product rights are protected, a company is unlikely to spend the estimated $200 million to support the research necessary to confirm a health benefit (O'Donnell, 1994). Since labeling regulations can restrict the ability of a manufacturer to make health claims directly on their products, other routes of promoting health

claims such as the use of magazine articles, advertising, and physician educational programs are commonly used to tout the nutritional benefits of food components.

In addition to problems with labeling regulations, incorporation of bioactive compounds into foods faces several challenges to the food processor. Incorporation of certain bioactive compounds could cause problems with the sensory quality of foods since these compounds would impart flavor (e.g. garlic and the astringency of phenolics) and color ( e.g. phenolics). Incorporation could also be difficult due to solubility, stability and detrimental effects on other food components. Solubility differences would be a major problem in the incorporation of fat soluble bioactive compounds (e.g. garlic derivatives, conjugated linoleic acid and certain phenolics) into low fat products. Stability of the compound must also be considered since many bioactive components such as lactic acid bacteria might not be able to survive the expected shelf-life of the product. Phenolics could also decrease the stability of other food components such as proteins and lipids since phenolics are known to cause protein precipitation and high concentrations of phenolics can accelerate the oxidation of lipids. These problems will limit the incorporation of many bioactive compounds into a wide variety of food products.

Before the challenges presented by current regulatory and food manufacturing issues can be fully addressed, more research is needed on the relationship between bioactive compounds and disease prevention, human safety, and the bioavailabilty of other nutrients. Most studies using bioactive compounds have centered on experimental animal models where extremely high does are studied. Very little is actually known about the effectiveness or even the absorption of bioactive compounds in humans. An additional problem with promoting bioactive compounds is their ability to prevent disease in one tissue while promoting disease in another such as the ability of green tea extracts to inhibit small intestine and promote liver cancer, simultaneously. Safety problems also exist since many bioactive compounds are in the form of extracts which could contain toxic as well as beneficial compounds. Caution should also be used since bioactive components can influence the bioavailability of other nutrients. For instance, phenolics decrease the absorption of proteins and minerals such as calcium and iron. Therefore their addition to food could actually have a negative impact on the nutrition of numerous populations. Since the scientific community has really only begun gathering evidence on the biological effect of bioactive compounds over the last 15-20 years, it is obvious that much more research is needed before the nutritional implications of these compounds is understood. While it may not be wise to dramatically increase the level of nonessential bioactive compounds in our diets, eating a well balanced diet containing a wide variety of food will naturally provide many of these potentially beneficial compounds.

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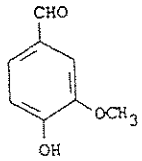
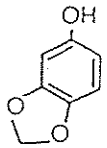
Figure 1: Chemical Structures of Several Phenolic Antioxidants




Vanillin

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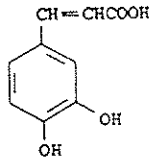
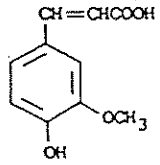
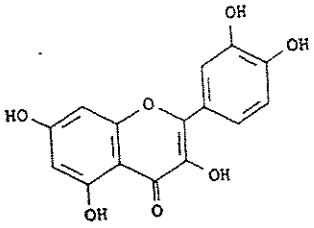


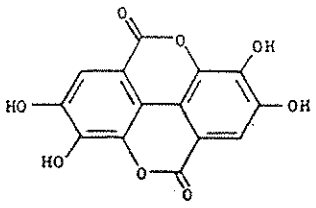

Caffeic Acid



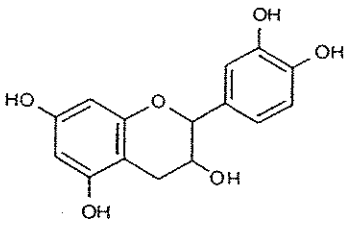

Ferulic Acid



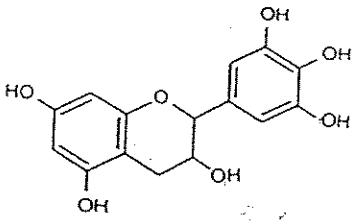

Quercetin



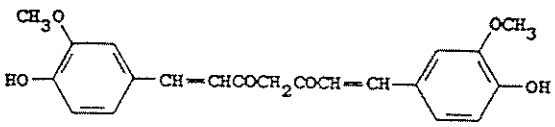

Ellagic Acid




Epicatechin

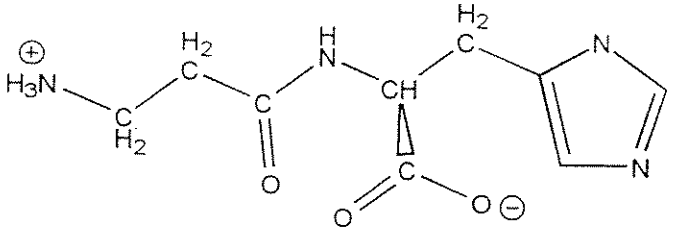



Epigallocatechin

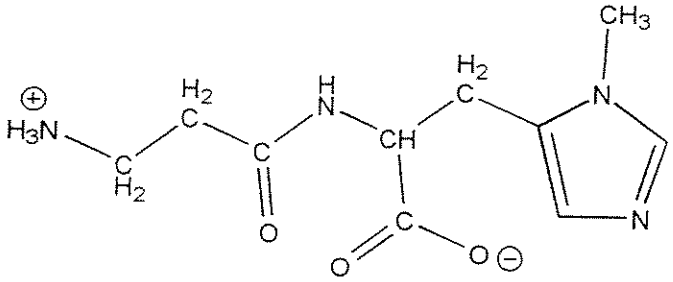



Curcumin

Figure 2: Chemical Structures of
Carnosine and Anserine



**CARNOSINE**



**ANSERINE**

# Carnosine–Vitamin E Study

## Mammary Cancer: Incidence

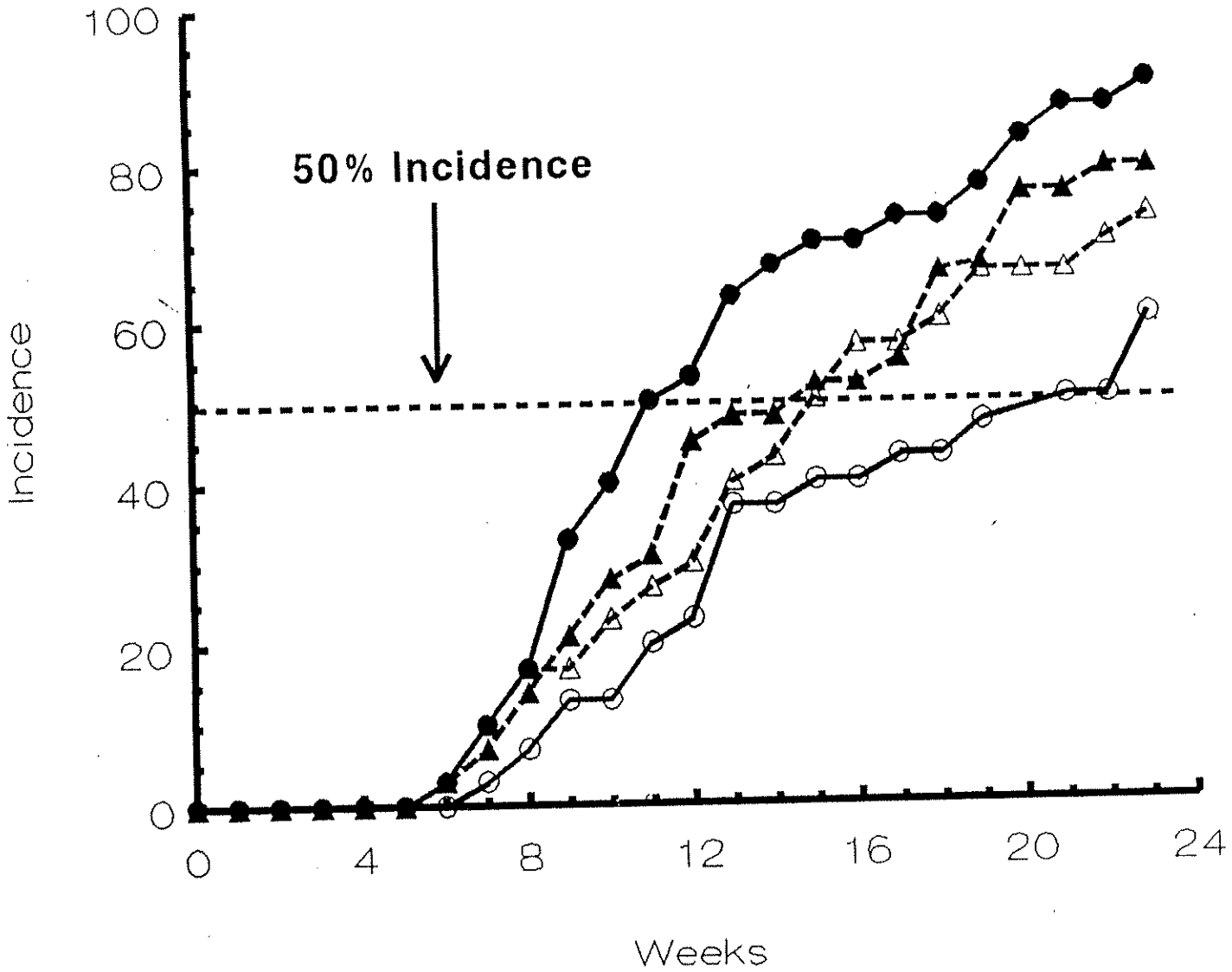



Figure 3: The Formation of Palpable 7,12-Dimethylbenz[a]anthracene (DMBA)-Induced Breast Cancer Tumors in Rats Fed Control Diets (No Supplements; -E,-C) or Diets Supplemented with a-Tocopherol Acetate (50 ppm; -E,-C), Carnosine (0.0875%; -E,+C) or Carnosine and a-Tocopherol (+E,+C).

Table 1. Examples of phenolic compounds in foods.

| Compound | Occurence |
|---|---|
| Vanillin | Vanilla beans, Cloves |
| Sesamol | Seseame Seeds |
| Caffeic Acid | Oats, Soybeans, Blueberries, Prunes, Grapes |
| Ferulic Acid | Oats, Soybeans, Blueberries, Prunes, Grapes |
| Quercetin | Tea, Coffee, Cereal Grains, Onions, Grapefruit |
| Epicatechin | Tea Leaves |
| Epigallocatechin | Tea Leaves |
| Ellagic Acid | Grapes, Strawberries, Raspberries |
| Curcumin | Tumeric, Mustard |

Table 2. Carnosine and anserine concentrations in various skeletal muscles (Crush, 1970).

| SOURCE | MUSCLE | CARNOSINE mg/100 g tissue (mM) | ANSERINE mg/100 g tissue (mM) |
|---|---|---|---|
| Chicken | Leg | 50 (2.9) | 167 (9.3) |
| *Gallus gallus* | Pectoral | 278 (16.4) | 983 (54.6) |
| Rabbit | | | |
| *Oryctolagus cuniculus* | Leg | 70 (4.1) | 400 (22.2) |
| Atlantic Salmon | | | |
| *Salmo salar* | | 0 | 400 (22.2) |
| Beef | | | |
| *Bos taurus* | Leg | 150 (8.8) | 50 (2.8) |
| Swine | | | |
| *Sus sp.* | Shoulder | 276 (9.2) | 20 (1.1) |
| Human | | | |
| | Quadriceps | 362 (21.3) | 0 |