

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Antioxidants and Natural Compounds in Mexican Foods

José Luis Silencio Barrita, Sara Montaña Benavides and Santiago Sánchez

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/61626>

Abstract

In Mexico there is a quality of climate and land suited to the cultivation and production of a wide variety of fruits and vegetables rich in natural antioxidants. Although these fruits and vegetables contain sufficient antioxidants, consumption is low, especially in at-risk populations such as children, pregnant women and the elderly. Fast food on the street and in restaurants is preferred to food at home, and more fruits are consumed than green vegetables. In virtually all social strata there is a marked preference for the consumption of fast food with a high content of saturated fat, salt, cholesterol, protein and simple sugars. The consumption of raw or cooked green vegetables has declined with at best the consumption of a serving a day when the World Health Organization (WHO) suggests at least 3–5 servings of vegetables a day. This decrease in the consumption of natural foods, and therefore in associated antioxidant components has been crucial in the development of chronic degenerative diseases such as obesity, cancer, diabetes and cardiovascular disease. Such diseases are the leading cause of death in the Mexican population according to data from the National Statistical Institute of Geography and Informatics (INEGI, 2013) [1]. On the other hand, there is an excessive consumption of food supplements containing these same natural antioxidants in a purer and more concentrated form than in natural food sources. Such supplements or additions were initially only consumed by athletes, but are now widely used by the general public without an understanding of the normal recommendations and possible toxic effects they may have on the user.

Keywords: antioxidants, Mexican foods, vegetables, fruits

1. Introduction

Although Mexico has a great variety of climates suited to the growth of a large amount of vegetables, their consumption is limited. National consumption is estimated at only a single serving of vegetable food per day, usually with the midday meal. The most widely eaten vegetable foodstuffs are: tomato, onion, lettuce, chili, green tomato, cucumber, avocado and

potato. Some vegetables are used as a condiment in the preparation of food (seasoning) and others as a spice, as in the case of vegetables such as coriander, parsley, epazote, mint and others. The average diet of the Mexican people also includes corn tortillas (maize) and wheat in the form of bread, which become "tacos" when the tortilla is used as a base on the one hand, and "tortas" when bread is used on the other. Added to these are – in raw form – lemon, onion, cilantro and chili. The chili is added in the form of sauces based on tomatoes, or cooked or roasted green tomato.

The quantities of these sauces that are eaten vary, which also makes it difficult to calculate the total amount of antioxidants consumed daily. A person can eat between 10 and 12 tacos – with an average weight of 50g each – per serving, or a cake whose weight can vary between 200 and 500g and whose filling is variable, but may include tomato and onion slices, as well as chili and avocado.

Most of these "tacos" and "tortas" are sold on the street as fast food, and are readily available and cheap [2]. This type of food is eaten between four and five times per week on average, either at breakfast, lunch or dinner.

This type of diet is not particular to any socioeconomic level and can be seen in any social stratum. Other foods included in the daily diet are legumes such as beans, peas, lentils and beans, consumed in large amounts in broths or soups, pasta and rice.

Due to this great variability and irregularity of content, consumption of antioxidants in the population is very low and many that are present are often removed by the effects of heat or frying.

The low consumption of antioxidants in fresh food correlates well with the presence of slowly evolving illnesses and chronic degenerative diseases, which are major causes of morbidity and mortality, as described by the health sector. They are leading causes of death and disease from stroke, cancer, diabetes and obesity.

In the last three decades, the daily consumption of fast food has increased and its preparation eliminates or reduces the amount of antioxidants present. This type of food is represented by pizzas, hamburgers, roast chicken and chicken type "crispí" for example.

Nutrition in Mexico is currently polarized since some areas enjoy a sufficient supply of food while others have an insufficient supply that have been promoted for many years [2]. However, even in areas where there is a good supply of food, consumption is often restricted to foods that promote the development of chronic degenerative diseases. Such is the case with the huge consumption of sugars and flours in foods that have contributed to the rise in metabolic syndrome in the past decade and have therefore triggered diseases such as diabetes. In addition to the increased consumption of sugars, are large amounts of saturated fatty acids (saturated fat) and salt which are promoters of obesity and hypertension, which further contribute to the current state of public health in Mexico. These same food combinations produce an organoleptic effect, which has become, unfortunately, the basis of the national diet. The consumption of nutrients that have a major impact on the overall health of the individual, such as some vitamins, many minerals and long-chain polyunsaturated fatty acids is neglected. Of the latter, omega-3 fatty acids (n-3, Morris system) are the least consumed by Mexicans.

The most important sources, as will be discussed later, are marine fish and seafood (algae). However, in Mexico, little fish is eaten. It is estimated that the per capita consumption of fish is lower than 10 g per year, and does not meet international recommendations. The WHO has recommended the consumption of at least three servings of fish per week to cover more than 50% of the current recommendation. Vegetables are another important source, but the consumption of vegetables is affected by their low general popularity [4].

Due to these considerations and the properties of n-3 fats, it is likely that there is a positive correlation between the prevalence of chronic degenerative diseases and a deficiency in their consumption. In addition, symptoms observed in diabetic, hypertensive and obese patients are probably better explained by the failure to consume this type of fat, and especially by the physiological, vascular, blood and cellular functions this molecules that express.

Chronic noncommunicable diseases (CNCD) are one of the biggest challenges facing the Mexican health system. There are several factors involved: the large number of cases, their increasing contribution to overall mortality, forming the most frequent cause of premature disability, and the complexity and high cost of treatment. CNCDs are a heterogeneous group of conditions contributing to death through a small number of outcomes (diabetes, cardiovascular diseases and cerebral vascular disease). The deaths are the result of a process begun decades earlier. The natural evolution of diabetes and cardiovascular disease can be modified by actions that change the clinical course of the conditions that determine its incidence. These include being overweight and obese, abnormal concentrations of blood lipids, hypertension, smoking, a sedentary lifestyle, improper diet and metabolic syndrome. This introduces opportunities for prevention, the development of prognostic tools and the creation of macro-economic models. And it is here where food, or the poor intake of foods, is linked to the high incidence and frequency of these chronic conditions. It is not only deficiencies in consumption that can lead to these clinical spectra, but also excessive consumption.

Variations in the prevalence of obesity, overweight, dyslipidemia, hypertension and metabolic syndrome in the period covered by National Health Surveys (1994–2006) are already documented. The percentage of the population with a higher weight than that desirable ($>25 \text{ kg m}^{-2}$, body mass index [BMI]) rose 13 % in the period from 1994 to 2000 and the change was even greater (33.5%) between the years 2000 and 2006. The same increasing trend was observed in the prevalence of **metabolic syndrome**, a concept that identifies patients with increased risk of developing diabetes or cardiovascular disease in the medium term. The percentage of adults with metabolic syndrome (defined by the criteria of the National Cholesterol Education Program) increased by 27.8% between 1994 and 2000 and 39.7% cases corresponded to individuals under 40 years of age. The data suggest that the contribution to the mortality of CNCD will increase in the medium term [5, 6, 7, 8].

The data suggest that the contribution to mortality by CNCD will increase in the medium term. The percentage of adults with diabetes (diagnosis established by a doctor) grew by 25% in 2006. It has been projected that 11.7 million Mexicans will have diabetes by 2025. Each national survey shows a rise (4.0 to 5.8% in the period 1994–2000) and from 5.8 to 7% from 2000–2012. Type 2 diabetes is a major cause of premature disability, blindness, end-stage renal failure and non-traumatic amputations. It is one of the 10 most frequent causes of hospitalization in adults.

The last link in the chain is evaluated by the effect of CNCD on mortality. The percentage of mortality attributed to outcomes associated with CNCDs has shown continuous growth. In just four years (2000–2004), the proportion of overall mortality explained by diabetes and cardiovascular diseases rose from 24.9 to 28.7% in men and 33.7 to 37.8% in women. As a result, since 2000 ischemic heart disease and diabetes have been the two most frequent causes of death in Mexico [9, 10, 11].

In relation to cancer:

Cancer is a disease with a major psychological, social and economic impact. According to a preliminary analysis by Delgado Gutierrez et al., between 2000 and 2010, there was an average of 66, 000 deaths annually in Mexico from cancer, equivalent to 13% of annual national mortality. From the total number of deaths from cancer, women represent – on average – 51% annually. The economically active population (aged 15–64) comprises 43%, and the population aged 65 years or older, 54%. During this period, lung cancer was the cause of highest mortality (6, 701) followed by malignant neoplasms of the stomach (5, 298) and liver (4, 819). Among cancers of the reproductive organs, prostate cancer generated the highest number of deaths (4, 690), followed by breast (4, 321) and cervix (4, 236). These six tumors caused 46% of the total number of deaths from cancer [12, 13].

There have been efforts to elucidate the number of new cases of cancer at the national level, by recording histopathology of malignant neoplasms (RHNM). However, these have been isolated and institutional and have not been systematized. The RHNM represented an initial effort through a hospital registry, but it ceased operation in 2002. The idea was for it to evolve towards a population-based register, whereby it would be possible to measure the real extent of the disease. The commitment is still pending. Due to the size of Mexico, the high prevalence of risk factors and the rapid aging of the population, it is necessary to create several regional registries that are population-based, inter-agency and that have access to uniform and systematic information enabling the recording, year after year, of all the new patients who receive a diagnosis of a malignancy. We know that up to four out of 10 cases of cancer are preventable, and that 30 patients could be cured or achieve disease control if their cancer is detected early and they are referred to receive optimal treatment [13]. In Mexico, efforts in primary prevention and early detection are poorly-organized and the lack of an adequate budget results in a lack of timely performance and quality. This largely explains why more than 70 cases of cancer are diagnosed only in the advanced stages, reducing the chance of recovery and at the same time generating the broad needs of palliative care to improve the quality of life of the patients.

1.1. Morbidity

Globally, it is estimated that in developed countries, three in four children with cancer survive at least five years after their treatment, but in developing countries about 60 will die in the same period (National System of Epidemiological Surveillance SINAVE, 2011). In general, younger children have better survival rates, which might be because there is less delay in diagnosis, treatment and its success among teenagers and young adults. This is due in part to the fact that young children have greater monitoring through their parents and the health

system, so any warning signs of developing disease, are noticed more quickly. In children, about five of the malignant tumors are related to hereditary mutations passed from parents to children, although not necessarily in all cases will the disease develop. An example of this is retinoblastoma, a cancer of the eye that occurs more frequently in children, and that parents identify through observing a whitish reflection in the eye of the child. In addition to the hereditary factor, there may be genetic mutations during fetal development that predispose individuals to cancer at early ages, as has been observed in leukemia, where one in every 100 children is born with a genetic anomaly and has greater risk, although only one in 8,000 develops the disease (National Cancer Institute, NCI, 2014). Signs and symptoms that allow early detection of cancers in the population under 20 years, include the following (Secretary of Health of the State of Veracruz, 2014): prolonged fever without an obvious cause; an increase in volume of any part of the body; weight loss; unexplained red spots or bleeding anywhere on the body, and general malaise [14].

It is important to note that symptoms will depend on the type of cancer, but the above are indicative of those symptoms requiring the child to be presented to the health services. In Mexico, analysis of hospital morbidity from cancer (patients who died in a hospital from the disease) during 2012 showed that in both men and women under 20 years of age, cancer of the hematopoietic organs was the leading cause of death, (59.5% in women against 58.8% in men). For males, the second leading cause of hospital discharge for cancer is the lymphatic system and related tissues (8.8%), followed by the brain and other parts of the central nervous system (6.3%). However, in women, the order is reversed with brain cancer and other parts of central nervous system the second commonest cause (6.7%) and the lymphatic system and related tissues the third (5.9%) [14, 15].

It is not uncommon to think that the high incidences of chronic degenerative diseases are correlated with a deficiency in the consumption of some nutrients that could be the cornerstone of the protection of the patient, and within these it is very likely that an imbalance in the consumption of an antioxidant and pro-oxidant, or its excess, that are the factors that trigger these diseases. Such is the case in the poor consumption of omega 3 fatty acids, selenium, zinc, iron, and vitamin C.

2. Antioxidants

Human beings are exposed to a large number of "oxidizing agents" such as pollution, stress, cigarette smoking and some other chemical compounds used in improvement or preservation of food. In addition, our body produces so-called "free radicals" (FR), which can cause oxidation of membranes and DNA damage, triggering a series of undesirable reactions that lead to diseases such as cancer, cardiovascular problems and aging. Antioxidants are compounds which, by their chemical structure, can slow the formation of free radicals and prevent or treat the above-mentioned diseases caused by oxidative stress. This oxidative stress is a consequence of environmental pollution, stressful jobs, indiscriminate consumption of

processed foods, excessive consumption of snacks and fatty foods and is encouraged by the presence of chronic diseases, self-medication and physical inactivity.

In order to quantify the antioxidant capacity of foods, the Oxygen Radical Absorbing (ORAC) test was developed. In this chapter, we show the concentrations of important antioxidants as measured in our laboratory, in raw and cooked foods, with a special emphasis on vegetables.

2.1. Micronutrients: Vitamins and minerals

In children under five years of age, dietary surveys indicate that the diet is energy deficient. The diet varies between 73 and 83% of needs, as well as having a low consumption of calcium, vitamin A, riboflavin and vitamin C (with averages of ingestion about 50% of recommended amounts). Although intakes of iron were greater than the dietary recommendations for other age groups, a large proportion came from corn and beans, foods that also contain high amounts of phytates and tannins, which inhibit the absorption of iron. This explains the presence of anemia in the population despite high intakes of iron. It should be noted that the methodology for collecting information of reminder 24 hours diet tends to underestimate the consumption of nutrients by between 15 and 20%, so it is possible that the deficiencies in energy and nutrients referred to were lower than reported. There were areas where the majority of the adult inhabitants were underweight, while in other areas, especially urban, there was a prevalence of being between 5 and 15% overweight. In particular, a group of workers in Mexico City was identified as 28% overweight. Also, the presence of anemia in women living in coastal areas was 20%. In rural regions and areas, the population had less variety in their diet, which was low in protein of animal origin and deficient in vitamins. The biggest nutritional problems were presented in the southern and southeastern regions of the country, followed by the downtown area, and to a lesser extent, on the coasts and the north of the country. Clinical signs of malnutrition and micronutrient deficiency are presented in severe conditions. Clinical signs of deficiency, such as hair pigmented and easy to boot (8%), scaly and pigmented skin (3%), alterations in the eyes (4.7%), smooth tongue (6.3%), cheilosis (6.6%) and edema (1.6%) have been documented in children. The latter is the most reliable sign for recognizing severe malnutrition. A presence of anemia was identified in 29% of preschool children, with 20% in rural and semi-rural areas, and 9% in urban areas.

3. Indicators of micronutrients

Anemia was determined using a portable photometer (HemoCue). In order to classify subjects with anemia, the following cut-off points of hemoglobin concentration in blood proposed by WHO were used: children aged 1–5 years, 110 g/L; children aged 6–11, 120 g/L; (non-pregnant) women aged 12–49, 120 g/L; (pregnant) women aged 12–49, 110 g/L; men from 12–14 years, 16 mg/dL; zinc deficiency is defined as serum zinc concentration of 65 ug/dL; deficiency of vitamin A as retinol serum 20 ug/dL; folate deficiency as concentrations of folate in red blood cells of 140 ng/mL; and deficiency of vitamin C, such as serum concentrations of Ascorbic acid at 0.2 mg/dL [13].

Anemia and deficiencies of various micronutrients are the commonest nutrition-based problems in Mexico, primarily affecting children and women of fertile age. In Mexico, the most important deficiency in terms of magnitude is that of iron in children of all ages (from 12 months to 11 years), which ranges from 36 to 67%. In women, the prevalence of iron deficiency is over 40%, though it is not the largest of the deficiencies observed in this population. The prevalence of low concentrations of vitamin C, an indication of low dietary intake of this vitamin, is very high and fluctuates between 30 and 40% in the case of children, and reaches 50% in women. The prevalence of low zinc consumption is around 30% in children under five years and women, with little more than 20% of those in school [13, 15]. Marginal vitamin A deficiency is between 20 and 30% in children and less than 5% in women. Folic acid deficiency ranges from 10 to 20% in children and 8% in women.

Studies of micronutrient deficiencies in the 1999 ENN survey showed that the most common is a lack of iron, mainly affecting rural areas of the country. The highest prevalence rates were reported in children under six years of age, while in schoolchildren and women of fertile age no physiologic load had a lower prevalence (INSP, 1999) [15].

In some cases, the consumption of vitamins in tablet form is recommended, but only justified if there is clinical evidence of its deficiency. Otherwise, it is best to consume natural food sources that contain the relevant vitamins. However, in Mexico, an almost magical power is given to vitamins in such a way that some sectors consume them without medical advice and in ignorance of official recommendations and potential toxic effects. They are recommended by stars of television, and used in gyms by personal trainers and the general public, and since a prescription is not required for their purchase they are within the reach of virtually anyone who can visit a hypermarket.

Treatments with vitamins such as folic acid, vitamins C, A, B₁₂ and B₁ are widely used by the medical sector for the specific conditions that require them. They are necessary for normal metabolism and even for the metabolism of other drugs that are prescribed.

4. Vitamin A

Vitamin A is a generic description covering all compounds which show biological activity of retinol, such as some retinoids and carotenoids (also known as pro-vitamin A). Retinoids are isoprenoid compounds normally found in animal and plant tissues. Because of this, vitamin A is found in different forms, but you can tell that there esterifies fatty acids, primarily as retinyl palmitate. It is likely that several forms of the vitamin are present in the same food, but in differing concentrations [16].

4.1. Vitamin A structure

The vitamin A molecule contains a β -ionone ring with an unsaturated side chain containing an alcohol group at the end of the chain, characterizing the main vitamer. (Fig.1)

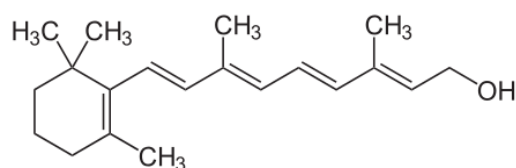


Figure 1. Structure of vitamin A (retinol)

4.2. Vitamer

Various forms of Vitamin A exist in food and are generally known as retinol alcohol (A_1). It is stored in tissues such as palmitic ester. The Isomer of retinol: 3-deshydroretinol (with 40% of the activity of A_1), is found in fish liver oils and has been shown to be converted to Vitamin A_1 [17] in rats.

The term vitamin A is broad as it includes all those compounds containing a β -ionone ring and designated as retinoids or derivatives of β -ionone, and that is why carotenoids are so called. The ring is very important as the activity of the vitamin depends on it. In fact, when the vitamin is exposed to sunlight, the ultraviolet light present in the sunlight breaks the ring and vitamin activity is lost. In vivo, retinol can be converted to its aldehyde, retinal, and this in turn can be converted to retinoic acid. This is an oxidation process wherein the alcohol (retinol) is oxidized to the aldehyde (retinal) and this in turn is oxidized to acid (retinoic acid). The reaction is thermodynamically irreversible and retinoic acid cannot be converted back to retinal, so the retinoic acid is rapidly oxidized and excreted. This action cannot be considered by itself as an example of an antioxidant reaction. However, the participation of vitamin E, beta carotene and vitamin C in the equilibrium of these reactions involving vitamin A is part of the antioxidant response. Vitamin A lies partly in cell membranes so that it is very likely be involved, like vitamin E, in free radical scavenging, itself becoming a free radical (possibly retinyl). This retinyl radical could purge, with vitamin C, free radicals seeking to initiate lipid peroxidation of the membrane. Retinol has been isolated from the retina of the eye and is the main form as vitamin works, in fact the only function biochemically proved, described by George Wald in 1967.

4.3. Sources

Vitamin A (retinol) can be ingested into the body or synthesized from plant carotenoids. Preformed vitamin A is present in animal tissues and the best sources are liver, milk, and the kidneys, where it is found in high concentrations in the form of fatty acid esters [18].

Vitamin activity occurs primarily in plant carotenes, which are precursors of vitamin A. Theoretically, a molecule of β -carotene should give rise to two molecules of retinol, however, under in vivo conditions this does not happen and only one molecule is obtained. Of the more than 100 carotenoids described so far, only 12 are converted to retinol and β -carotene is the most important of these. As a result, efficiency of the conversion of carotenoids in retinol is 1/8 (i.e. 100/12). Therefore, the amount of vitamin A from plant foods should be one-eighth of the concentration of the carotenoids present in the food. Of course this may change and the amount

could increase or decrease, depending on how many carotenoids or β -ionone derivatives are contained in foods. Thus, it is often recommended to ingest some retinol equivalents from a diet containing both animal and vegetable foods [19].

This situation is the result of inefficiency in the absorption of β -carotene from vegetables and fruits, its chemical structure, biological activity and the dynamics of oxidation of retinal to retinoic acid, which is quickly removed.

Table 1 shows the main sources (with highest concentrations) of vitamin A, although in Mexico there are other widely consumed sources with lower concentrations.

	Food	Content (UI/100g)
1	Dehydrated carrot	100,000
2	Cod liver oil	85,000
3	Dried red pepper	77,000
4	Roasted lamb liver	74,500
5	Homemade paprika	60,604
6	Fried beef liver	53,400
7	Dehydrated sweet potato flakes	47,000
8	Red pepper	41,610
9	Chile powder	34,927
10	Fried beef liver	32,700
11	Canned pumpkin	27,383
12	Parsley dried	23,340
13	Dehydrated alfalfa	22,940
14	Raw red pepper	21,600
15	Liver cooked turkey simmered	17,500
16	Liver cooked chicken simmered in grill or skillet	16,375
17	Canned carrots, drained	15,000
18	Fried pork liver in margarine	14,900
19	Dried peaches, sulfur, crude	14,100
20	Green dandelion, raw	14,000
21	Carrots, raw	11,000
22	Green, raw mustard	9,900
23	Spinach, raw	8,100
24	Sweet potato shell, cooked	8,100
25	Beef cooked in water	7,800
26	Green radish, raw	7,600
27	Swiss, raw chard	6,500
28	Peanut butter	6,400
29	Dried peaches, raw	5,000
30	Cantaloupe, raw	3,400

Food		Content (UI/100g)
31	Margarine	3, 307
1 UI =0.300 ug retinol		

Table 1. Sources of Vitamin A

4.4. Functions

Preformed vitamin A is present in the diet as retinyl esters. Esters of vitamin A are hydrolyzed during digestion by pancreatic hydrolases, absorbed in their free form and re-sterified with fatty acids within the intestinal mucosa, entering the circulation by passive diffusion with chylomicrons formed. Beta-carotenes from plants are substrates for the synthesis of vitamin A, obtaining retinaldehyde, which is then reduced by a specific enzyme called aldehyde reductase (using NADH as a cofactor), and converted to retinol. Retinol from any source (animal or plant) is stored as retinyl esters in the parenchyma of the liver. The normal body retinol reserve varies from 300–900 mg (1 IU=0.300 ug trans-retinol). Prior to release, esters in the liver are hydrolyzed and the free alcohol binds to a specific binding protein retinol binding protein (RBP), which transports it to peripheral tissues. This protein is a single polypeptide chain of 182 amino acid residues with a molecular weight of 21 KD. It is synthesized initially as a 24 KD pre-RBP by parenchymal cells, and also by post-translational modification of a polypeptide removed (3.5 KD). Secretion is hormonally regulated by circulating estrogens and the same concentration of the vitamin. In healthy individuals, the concentration of RBP in plasma is equal to 40–50 ug/mL. This protein is catabolized in the kidney, however catabolism is reduced by the binding of the protein to retinal [20, 21].

The best known function of vitamin A is its role in vision, in which the prosthetic group is a series of carotenoid proteins providing the molecular basis for visual excitation [22, 23]. The photosensitive chromophore group responsible is 11-cis-retinal, present in a group of visual pigment cells of the eye known as "rods" and "cones". The latter have precision and accessory pigments at wavelengths with maximum absorption peaks in the red, green and blue parts of the spectrum. The "rods" have precision too and contain the pigment **rhodopsin** and cones contain the pigment **iodopsin**. In any case, 11-cis-retinal is bound to a lysine residue of a specific protein called opsin.

When light strikes these molecules, there is a light-induced isomerization transforming the 11-cis-retinal to the all-trans-retinal form. This reaction results in the dissociation of the protein-vitamin complex. This dissociation is coupled to nerve centers in the brain that respond to vision stimulation, so that each nerve impulse caused by each of the breaks of rhodopsin molecules produces an image point. The union of all these points in the brain leads to the display of an image. This complex and efficient phenomenon occurs millions of times per second each time we open our eyes. Thus we can say that the process of vision is a cyclical process as its constituents thereof are reconstituted. The all-trans retinol can convert enzymatically in the dark to the cis form, which is then esterified and stored, presumably among the lipids of epithelial cells. Rhodopsin regeneration includes the hydrolysis of retinyl esters

to produce 11-cis-retinol which is transported (via RBP) to the outer part of the stick cell which is then oxidized to 11-cis-retinal [24].

The "extra-retinal" functions are less important than the visual function and are poorly understood. They are collectively referred to as vitamin systemic functions as they use retinoic acid more effectively than retinal, and secondly their biochemistry is very different and even more complex. Vitamin A is also involved in cell differentiation, morphogenesis, transmembrane transport and the immune response. A significant portion of absorbed vitamin A (40%) is oxidized or conjugated in the liver and secreted in bile through feces. Retinoids apparently affect cell differentiation in a manner analogous to steroid hormone action, by binding to nuclear chromatin for transcription processes signals. It has been proposed that with retinoic acid they synergistically stimulate the production of thyroid hormones and growth hormone in cultured pituitary cells [25, 26].

Its role in cell differentiation is based also because it shares regions of binding to ligands and DNA along with vitamin D₃, steroid hormones and thyroid hormones (T₃). These ligand-receptors for retinoic acid (RAR α and RAR β) bind to promoter regions of specific genes activating transcription. It is also proposed that vitamin A plays a co-enzymatic role, as a carrier of sugars in the synthesis of glycoproteins, whose functions at the cell surface are adhesion, aggregation, recognition and other interactions.

Vitamin A is necessary for reproduction, but the biochemical basis for this is unknown. Experiments in rats indicate that retinal is more responsible for these actions in mammals than retinoic acid. Rats subjected to retinoic acid grow well and look healthy, but are unable to reproduce, and females are unable to get pregnant, abort and reabsorb their products while males show damage to the spermatogenesis process. Injections of retinol into the testes restores spermatogenesis, indicating their direct role [27].

Because vitamin deficiency results in damage to the differentiation of epithelial cells without damage to the proliferation, its role in the etiology of epithelial tumors, including carcinomas is questioned. In fact, squamous metaplastic changes seen in vitamin deficiency are similar to experimentally-induced precancerous lesions. Therefore it is proposed that retinol – as an anticarcinogen – acts against the high levels of carcinogens in tumors and prevents the expression of malignant phenotypes. Thus the hypothesis that the nutritional status of vitamin A can increase or decrease the risk of cancer is supported.

Although it also is not very clear, there is support for the idea that vitamin A is important in immunocompetence. Vitamin deficient animals are more susceptible to infection than those with a sufficient intake of the vitamin. Epidemiological studies indicate that a state of deficiency is closely related to a higher incidence of disease and increased mortality. Vitamin deficiency affects immunity through different routes. Retinoids act on the differentiation of immune cells, increasing lymphocyte mitogenesis and phagocytosis of monocytes and macrophages. Carotenoids affect the survival of activated NK cells and T helper cells, modifying the release of cytosine-type products from activated lymphocytes and monocytes [28].

4.5. Deficiencies

Liver and tissue levels of vitamin A compensate for low intakes of it in countries like Mexico. The speed with which the body's stores are mobilized, depends on the severity of the deficiency and how fast are released it between different tissues. Because the vitamin is present in different organs, a deficiency will include many nonspecific signs. One of these signs is related to vision, and there are ocular signs such as nyctalopia and xerophthalmia. The first of these responds quickly to therapy with the vitamin, while the second disorder involves permanent changes to the eye, which cannot be corrected. However, in cases of xerophthalmia, timely therapy can disrupt early-stage lesions before they become permanent injuries [29].

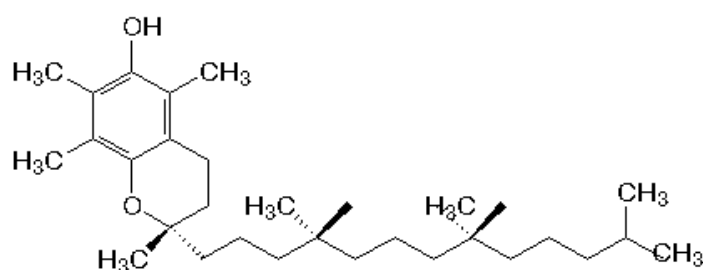
Vitamin A levels are decreased in night blindness, childhood blindness, hypothyroidism, infertility and teratogenesis, liver disease, xerosis, chronic infections, and pyrexia. A concentration lower than 10 mg of vitamin A/dL serum is indicative of severe hepatic impairment and absence of reserves. It also decreased in disseminated tuberculosis, carcinoid syndrome (very low) protein malnutrition, alpha-beta-lipoproteinemia and cystic fibrosis. Nyctalopia (night blindness) is a condition in which objects appear clear by day but are unclear at night. Poor dark adaptation is the best sign of vitamin A deficiency [30].

5. Vitamin E

5.1. Definition

Vitamin E is defined as all those forms exhibiting activity of α -tocopherol, which is the main representative of this group of compounds. The α -tocopherol is a derivative of phytyl and trimethyl hydroquinone alcohol.

The basic structure of this vitamin is chromanol ring and a phytyl tail in the case of tocopherols, and an unsaturated tail in the case of tocotrienols. See Figure 2.



Vitamin E (α -tocopherol)

Figure 2. Vitamin E structure

Also known as tocopherol (α -tocopherol is the most powerful and abundant antioxidant in foods). These compounds are potent fat-soluble antioxidants that protect the integrity of cell

membranes (fig 3) and are associated with rich sources of vegetable oil. If no free vitamin E is present, free radicals oxidize fatty acids of the cell membrane and thus affect cellular structure [31]. This is the basis for the suggestion that Vitamin E important in preventing both aging and cancers. Vitamin E increases the absorption of vitamin A because it prevents oxidation in the intestine. In addition, its antioxidant action increases in the presence of zinc. In foods, vitamin E is found mainly in vegetable oils, wheat germ oil seeds (walnuts, peanuts, almonds, etc.), and green leafy vegetables, etc. Absorption is relatively poor (20–80%) and is linked to dietary lipids. Vitamin E is stored in liver and adipose tissue and is absorbed through micelles. Its use depends on the presence of adequate fat and pancreatic and biliary function. Once absorbed, it is captured by chylomicrons and very low-density lipoproteins (VLDL), which are found in the lymph. Vitamin E stored in the liver is subsequently reattached to VLDL and HDL, to be taken up by cells and is mainly directed to lipid membranes. It is excreted primarily via the bile and feces, and to a lesser extent in the urine [32].

5.2. Vitamin E sources

Vitamin E is found in large quantities in plants. It is present in high concentrations (0.1–0.3%) in wheat germ, maize, sunflower seed, rapeseed, soybean, alfalfa, and lettuce. Alpha-tocopherol is usually found naturally as beta and gamma tocopherols. The pure form of the vitamin is a pale yellow viscous oil [33].

	Food	Content (mg/100g)
1	Wheat germ oil	149.4
2	Sunflower seed oil, 60% linoleic acid and higher	44.9
3	Almond oil	39.2
4	Cottonseed oil	35.3
5	Oil, linoleic acid above 70%	34.1
6	Alfalfa seeds	33.0
7	Rice bran oil	32.3
8	Margarine, salt, liquid oil	28.4
9	Dry almonds, with entire deck	27.9
10	Whole hazelnuts, shelled	21.0
11	Cod liver oil	20.0
12	Palm oil	19.1
13	Corn oil	14.3
14	Sunflower seed essence, dry shell	13.0
15	Olive oil	11.9

	Food	Content (mg/100g)
16	Peanut oil	11.6
17	Soy oil	11.0
18	Fat, vegetable	9.9
19	Roasted peanuts in shell, whole and savory	9.7
20	Wheat, soya blend and wheat flour	7.0
21	Commercial chips	6.4
22	Canned tuna in oil	6.3
23	Black, raw moras	3.5
24	Asparagus, boiled	2.5
25	Porridge	2.3
26	Green radish, raw	2.2
27	French fried shrimp, seasoned with breadcrumbs and batter	1.9
28	California avocado, raw shell	1.7
29	Rye, whole grain	1.7
30	Butter, regular, salt	1.6

Table 2. Sources of Vitamin E

5.3. Functions

Vitamin E is absorbed from the gastrointestinal tract by a mechanism similar to other fat soluble vitamins, and it enters the blood initially via associated lymph chylomicrons, and then with lipoproteins, which correlates very well with the concentration of vitamin E and plasma lipid levels. It is stored in many tissues, residing mainly in the non-polar fraction of the lipid membranes in the center of the lipid bilayer. This feature protects against a body storage deficiency for long periods and is a basis for operation [34].

Vitamin E functions as an important natural antioxidant that prevents the spread of damage caused by free radicals to biological membranes. An important consequence of deficiency is that subjects suffer from anemia and peripheral nephropathy. The vitamin is an excellent "scavenger" of peroxy radicals and preferentially protects polyunsaturated fatty acids (PUFAs) in the phospholipids of biological membranes [35].

When lipid hydroperoxides are oxidized to peroxy radicals (ROO^*) these react 1000 times faster with vitamin E (VIT E-OH) than PUFAs (RH). The hydroxyl group of vitamin E reacts with an organic peroxy radical to form an organic hydroperoxide and the tocopheroxyl radical (VIT EO^*). Hence vitamin E has become a free radical preventing autoxidation membrane lipids. See Figure 3.

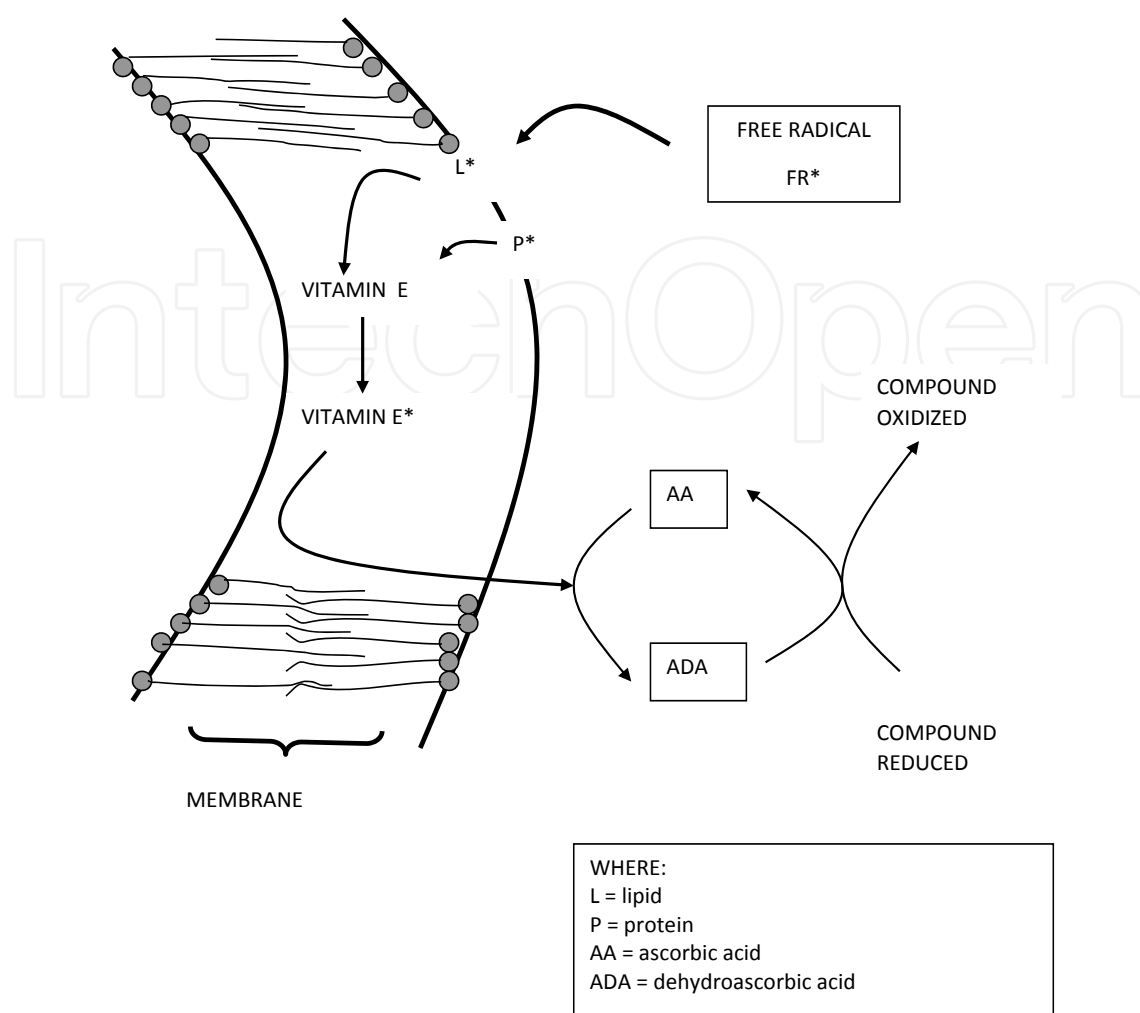


Figure 3. Functions of vitamin E

The formed tocopheroxyl radical expose its electron outside the lipid bilayer, into the aqueous part, to receive a part of any hydrogen donor reducing, such as vitamin C, which accepts and donates the electron hydrogen from vitamin E, being this in reduced form. This replenishes the vitamin so that its function becomes cyclical. Other compounds have been demonstrated to regenerate vitamin E and eliminate the symptoms of vitamin deficiency. These are selenium, glutathione, ubiquinone and other thiol groups (some amino acids with sulfhydryl groups such as cysteine). Thus the antioxidant activity of the vitamin depends not only on its concentration, but also on antioxidant compounds present in the intra- and extracellular water phase. The antioxidant status will also include the correct function related to maintaining an optimal state of oxide reduction for cell enzymes, which are also important activities of the enzymes catalase, glutathione peroxidase and superoxide dismutase.

Cells use superoxide dismutase, catalase and glutathione peroxidase to protect against reactive oxygen species. At the same time the body is careful to keep all iron and copper ions bound on protein transport or storage. Thus there is approximately three to four times more iron binding capacity in transferrin than in plasma, so there should be no free iron ions. Combining

elements such as iron with transferrin protein cannot stimulate lipid peroxidation and the generation of hydroxyl radicals, and this is also true for copper bound to albumin or ceruloplasmin.

All of these are regarded as primary defense mechanisms against free radicals, but there are also secondary antioxidant defenses and alpha tocopherol is the most important of these because it acts as a chain-breaking antioxidant. Vitamin E is a lipid soluble molecule, found in biological membranes or LDL, and possesses a hydroxyl group whose hydrogen is easily removed. When alkoxy or peroxy radicals are generated during lipid peroxidation, these combine preferentially with the antioxidant rather than an adjacent fatty acid.

The alpha tocopherol chain reaction ends and a new free radical is formed, which is less reactive and attacks the adjacent fatty acids less aggressively. There is evidence that this radical tocopherol migrates to the surface of the membrane and becomes alpha tocopherol through reaction with ascorbic acid. Therefore, vitamin C and alpha tocopherol help to minimize the effects of lipid peroxidation in LDL and membranes (see Figure 3).

5.4. Deficiencies

Vitamin E deficiency is very rare in humans and is unlikely to be of dietary origin due to the wide distribution of vitamin E in many foods. However, deficiency can occur due to genetic abnormalities such as a deficiency in the synthesis of tocopherol transfer protein, malabsorption syndromes of fats as seen in children with chronic cholestasis, and pancreatic insufficiency in cystic fibrosis, short bowel syndrome, steatorrhea chronic and total parenteral nutrition [36, 37, 38].

The primary manifestations of vitamin E deficiency include areflexia, ataxia, myopathy and pigmentary retinopathy. Hemolytic anemia develops in children if they are fed with a diet low in vitamin E during the first eight weeks of life. Vitamin E deficiency in chickens causes a generalized exudative diathesis, limited eye movement, and possibly paralysis.

5.5. Requirements and recommendations

The requirement decreases if the diet is high in polyunsaturated fatty acids and antioxidants. It has been estimated that the minimum daily requirement of tocopherol is 3–4 mg/day. However, it is known that 7.3 mg eq tocopherol/day is required to reduce the peroxidation of membrane lipids [39]. The daily recommended intake is 10 mg for men and 8 mg for women. The recommended daily amount (RDA) is contained in 5 mL of polyunsaturated oil.

6. Vitamin C

A diet including a sufficient intake of fruit and vegetables is almost always associated with beneficial health effects and a decreased risk of disease. All fruit juices are beneficial, particularly citrus juices, with orange, lemon, tangerine and grape being the best sources of vitamin C. The whole of Mexico is suitable for the cultivation of these citrus fruits, and chili peppers,

which also have a high Vitamin C content. Studies to date, indicate that vitamin C and fruit juices rich in vitamin C improve blood lipid profile, reduce oxidative stress and prevent atherogenic modification of LDL and platelet aggregation, in addition to improving HDL levels. Vitamin C has many important roles in health, ranging from its effects on the immune system to its intra- and extracellular molecular effects [39, 40].

Ascorbic acid (AA), commonly known as vitamin C, plays an important role in the human body, although its function at the cellular level is not yet clear. It is necessary for the synthesis of collagen, a protein that serves many functions in the body's connective tissues. Among the substances and structures containing collagen are bone, cartilage and surrounding material, as well as excipients and bonding materials between muscle, skin and other tissues. AA is also required for the synthesis of hormones, neurotransmitters and the metabolism of certain amino acids and vitamins. It is also required in the liver for the detoxification of toxic substances and in the blood for immunity. As an antioxidant, histamines and AA react with peroxides to reduce inflammatory symptoms [41].

Its antioxidant capacity is associated with reduced incidence of cancer. The requirements of vitamin C for adults are well defined, but these have not been uniform across different cultures, so its need has been defined in a culture-specific manner.

Some epidemiological data have stated its usefulness in reducing colds through increased consumption of foods rich in the vitamin, and people sometimes consume an "overdose" of it. Most reports mention that slight increases in levels of vitamin C in the blood reduce the risk of death in all conditions. Although vitamin C has many functions, its role in health is almost always discussed in relation to its function as an antioxidant and its effects on cancer, blood pressure, immunity, drug metabolism, and the urinary excretion of hydroxyproline [42].

Antioxidants have important roles in cell function and have been implicated in processes associated with aging, vascular disease, inflammatory damage and cancer. In its antioxidant role, AA is useful because it contributes to the maintenance of the vascular system and the reduction of atherogenesis through regulation of collagen synthesis and the production of nitric oxide and prostacyclin. In addition to an antioxidant role, AA acts at the molecular level as a cofactor for enzymes such as dopamine hydroxylase (EC 1.14.17.1), influencing the concentration of neurotransmitters, improving lysosomal protein degradation and mediating the consumption of monosodium glutamate (MSG) [43].

6.1. Sources

Vitamin C is one of the main constituents of fruits and vegetables, which also contain citric acid, oxalates and substances such as anthocyanins, carotenes and dyes that make it difficult to quantify amounts present when colorimetric methods are used.

There is currently a great interest in the consumption of natural foods, particularly with respect to the nutrient content of fruits, vegetables and vitamin C. This interest is due in part to the widespread use of vitamin C in the food and pharmaceutical industries. It is used as a supplement, additive, conservative, and antioxidant in processed foods. Table 4 shows the main foods that are good sources of vitamin C [44].

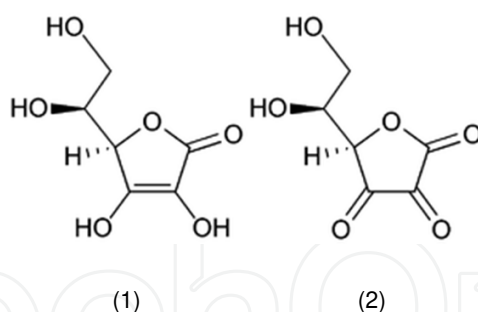


Figure 4. Structure. (1) Ascorbic acid and (2) dehydroascorbic acid

6.2. Requirements and recommendations

The recommended daily intake needed to avoid the appearance of symptoms of diseases caused by deficiency of vitamin C is 60–100 mg. Infants require a little over 100 mg / day. There is controversy over the required minimum amount of this vitamin. We must take into account that this vitamin is very heat labile in the presence of oxygen. Ascorbic acid is used in the treatment of scurvy, with dosage requirements best measured by determining the urinary excretion after a loading dose. Depending on the rate at which saturation is required, the recommended daily dose ranges between 0.2–2.0 g/day. In cases of vitamin C deficiency, tissue saturation is achieved with three daily doses of 700 mg c/u, over three days. Harris defined saturation of Vitamin C as sufficient storage in tissues so that excretion of 50 mg or more of ascorbic acid occurs in a period of 4–5 hours after a dose of 700 mg/day [45].

Decreased levels of vitamin C in smokers are explained by consumption of fewer sources of the vitamin. In smokers, 50% more than the recommended dose of vitamin is required. Because the RDA is defined as the average daily intake that is sufficient to meet the nutritional requirements of healthy individuals in a population group, it is necessary to continually reassess recommendations for vitamin C. All studies concerning recommended intake, suggest that 90–100 mg is sufficient for maximum reduced risk of chronic disease in non-smoking men and women, although some reports suggest amounts of up to 120 mg/day [46].

6.3. Antioxidant activity

Vitamin C is an important water-soluble antioxidant present in biological fluids. An antioxidant is defined as "any substance that when present at low concentrations compared with those of the oxidizable substrates (e.g., proteins, lipids and carbohydrates and even nucleic acids) significantly delays or prevents oxidation of that substrate." The definition given by the Panel of Dietary Antioxidants and Related Compounds in the Food Nutrition Board is that "a dietary antioxidant is a substance in foods that significantly decreases the adverse effects of reactive oxygen species (ROS), reactive nitrogen species (RNS) or both, on normal physiological function in humans."

Vitamin C rapidly clears reactive oxygen and nitrogen species such as superoxide, hydroperoxyl radicals, aqueous peroxy radicals, oxygen "singlet", ozone, peroxy nitrite, nitrogen

dioxide, nitroxide radicals, and hypochlorous acid, so it effectively protects other substrates from oxidative damage [46, 47].

Although AA reacts rapidly with hydroxyl radicals (constant speed $> 10^9 \text{ L mol}^{-1} \text{ s}^{-1}$) it struggles to purge this radical preferentially over other substrates. This is because the hydroxyl radicals are very reactive and so combine with any substrate in their immediate environment, close to a speed limited by diffusion. Vitamin C may also act as a co-antioxidant to regenerate α -tocopherol (vitamin E) from α -tocopheroxyl radicals produced when purging liposoluble radicals. This is a potentially important role because in vitro experiments have shown that α -tocopherol can act as a pro-oxidant in the absence of a co-oxidant such as Vitamin C. However, the in vivo interaction between the two vitamins is not yet clear. AA can regenerate urate, glutathione, and β -carotene in vitro from their oxidation products with an unpaired electron (urate radicals, glutathionyl radicals, and cations of β -carotene radicals) [48, 49].

Two important properties of vitamin C make it an ideal antioxidant. The first is its low ascorbate reduction potential (282 mV) and its oxidation product with an electron, the ascorbyl radical (2174 mV), which is derived from the functional group *en-diol*. These low reduction potentials of ascorbate and the ascorbyl radical, are potentially suitable for redox reactions, which is why vitamin C acts as a water soluble terminal antioxidant molecule. The second property that makes it an ideal antioxidant is its stability and the low reactivity of ascorbyl formed when the ascorbate radical purges reactive oxygen and nitrogen species.

Food	Concentration (mg / 100 g EP)	Moisture (%)
Squash	7.2±0.9	94.2
Spinach	8.5±0.2	91.6
Potatoes	74.6±6.7	79.7
Cucumber	93.0±7.1	96.0
Green tomato	222.8±10.7	90.9
Poblano chile	191.0±7.8	91.9
Green pepper	195.5±9.5	94.3
Nopales	268.9±30.1	95.2
Cambray onion	17.0±0.7	93.0
Carrot	50.4±5.6	87.2
White cabbage	184.7±17.2	93.2
Grapefruit	261.3±10.7	87.8
Mango	319.6±5.3	84.6
Watermelon	56.2±8.9	92.5
Banana	333.7±6.3	76.5

Food	Concentration (mg / 100 g EP)	Moisture (%)
Orange	279.8±39.4	84.0
Mamey	31.6±1.2	66.8
Plum	331.1±17.9	88.1
Grape	66.1±13.6	84.3
Apple	458.1±17.8	84.5
Beet	70.8±6.8	85.3
Lemon	39.4±2.5	88.3
Avocado	256.2±61.9	84.3
Sweet lime	306.8±23.4	89.8

Table 3. Content of ascorbic acid in Mexican fruits and vegetables [49]

EP= edible portion

7. Phenolic acids and polyphenolic compounds

Phenolic compounds are a large family of nutraceuticals that have beneficial health properties, ranging from inhibiting the spread of cancer, prevention of atherosclerosis, strokes and heart attacks, and inflammation. They are divided into three groups:

a) Isoflavones: a group of compounds present in some vegetables, especially soybeans. Within this family of isoflavones are daidzein, glycitein and genistein. Isoflavone intake plays an important role in reducing the risk of disease. Its antitumor, anticancer, antioxidant and immune response-enhancing actions have been shown. It also important in reducing cardiovascular risk and symptoms associated with effects of the menopause. Isoflavones exist in foods in their conjugated form. When ingested, these conjugated isoflavones undergo hydrolysis by β -glycosidases in the intestine to produce bioactive aglycones (daidzein and genistein). These can absorb or metabolize much more than other specific metabolites such as equol. The further metabolism of aglycones appears to be strongly influenced by diet. When in a high carbohydrate environment, increased intestinal fermentation occurs, resulting in more isoflavones being transformed into equol. This is relevant because the potency of equol is higher than its precursor daidzein. Intestinal bacteria also have an effect on the metabolism of isoflavones. When the intestinal flora is low (e.g. with the use of antibiotics in newborn babies) metabolism also falls. In germ-free animals it has been shown that isoflavones are not present in blood or bile. As endogenous estrogen (estradiol), isoflavones are metabolized in the gut and liver. Absorption occurs throughout the intestine and in secreted bile and urine. The excretion of isoflavones can vary greatly between individuals, being influenced by the fact that

each person has their own specific intestinal microflora. Once absorbed, equol shows less affinity is limited to whey proteins and thus has a greater availability than estradiol [50, 51].

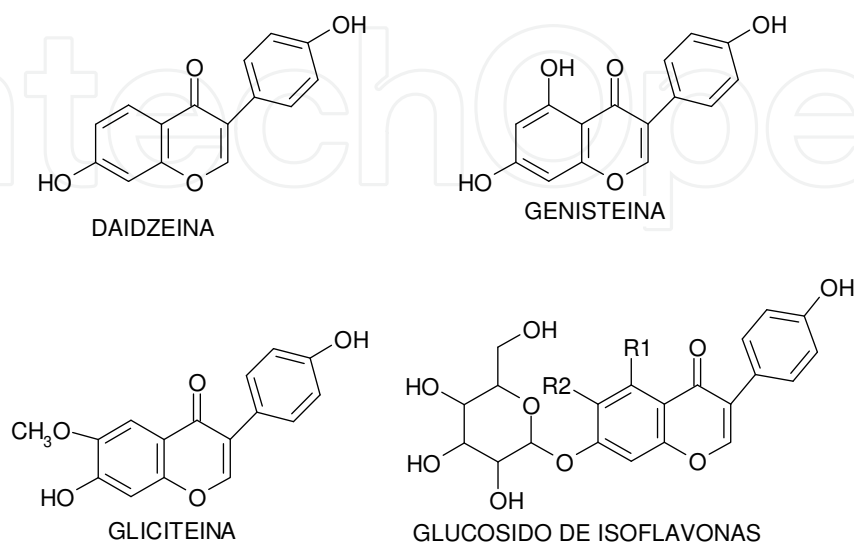


Figure 5. Isoflavones structure

b) Flavonoids and anthocyanins: Flavonoids and anthocyanins comprise a diverse class of pigments, which are normal found in cells or tissues that impart color to food. Flavonoids are similar to anthocyanins, which are water soluble vacuolar pigments present in the fluid in cells responsible for the majority of red, blue and violet flowers and leave the chemical structure colorations. Approximately 800 flavonoids are known and their number is increasing rapidly. One of the main groups is the flavonols: kaempferol, quercetin and mircetina. Another, less common group, is the flavones, comprising apigenin, luteolin and tricetina. In fruits, they are mostly found in the pericarp, so it is best to ingest the fruit unpeeled but washed properly beforehand. It is also important to note that many of these compounds are found in varying proportions in different types of wine, accounting for the preventive effect of moderate wine consumption on cardiovascular disease, cancer, and other degenerative diseases. Ingestion is recommended to maintain healthy tissues and promote a proper balance of hormones and antioxidants in the body, as many flavonoids appear to have an important role in human nutrition, and present very interesting medicinal properties, such as being antioxidant, anticancer, antithrombotic and reducing blood cholesterol levels [28].

c) Phytochemicals such as phytosterols, Phytosterols: plant sterols are widely distributed in nature and have a structure very similar to cholesterol. It has long been known that these sterols have a hypocholesterolemic effect when ingested in the range of 1–3 g/day, and are considered as important factors in preventing cardiovascular disease. Consumption is recommended for individuals with mild or moderate hypercholesterolemia.

Phytosterols; structures, main sources and importance

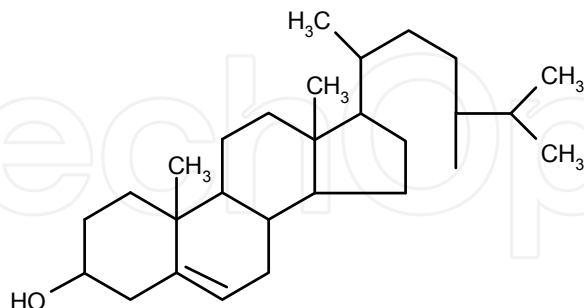


Figure 6. Campesterol or 24 α -methyl-5-cholesten-3 β -ol, 24 α -metilcolesterol, 24(R)-metilcolesterol, (24R)-5-ergosten-3- β -ol. Chemical formula $C_{28}H_{48}O$, MW 400.68 and melting point of 157–158°C. [52, 53]

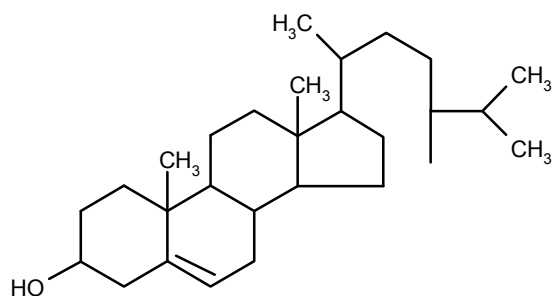


Figure 7. Sitosterol known as β -sitosterol, estigmast-5-en-3 β -ol, 24- β -etilcoles-5-en-3 β -ol. Chemical formula $C_{29}H_{50}O$, MW 414.71, mp 140 °C. [52, 53]

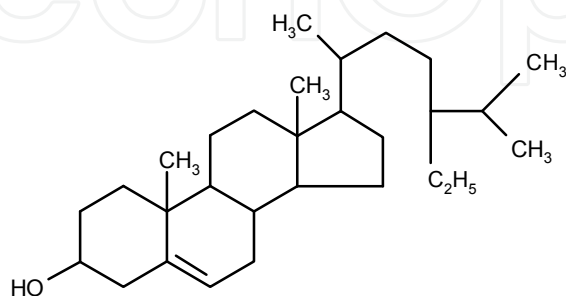


Figure 8. Stigmasterol, also called (22E)-estigmasta-5, 22-dien-3 β -ol or 24 α -etilcolesta-5, 22-E-dien-3 β -ol. Chemical formula $C_{29}H_{48}O$, MW 412.69, mp 170°C. [52, 53]

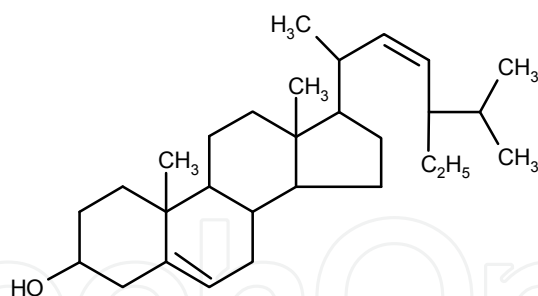


Figure 9. Fucosterol, also known as [24 (28) E]-estigmasta-5, 24 (28)-diene-3 β -ol, [24 (24') E] -estigmasta-5, 24 (24') -dien-3 β -ol or 24E-etilidenecolesta-5, 24 (28) -diene-3 β -ol. Chemical formula $C_{29}H_{48}O$, PM 412.69. [52, 53]

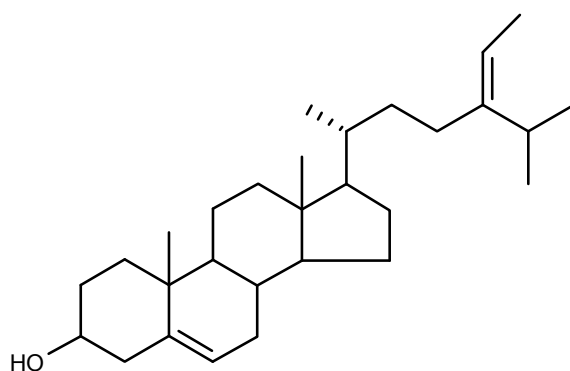


Figure 10. 5-avenasterol (5-avenasterol), called isofucosterol, 28-isofucosterol, 29-isofucosterol, 24Z-etilidenecolesta-5, 24 (28) -diene-3 β -ol, [24(28Z)] -estigmasta-5, 24 (28) -diene-3 β -ol or [24(24')-Z]-estigmasta-5, 24(28')-diene-3 β -ol, chemical formula $C_{29}H_{48}O$, PM 412.69. [52, 53]

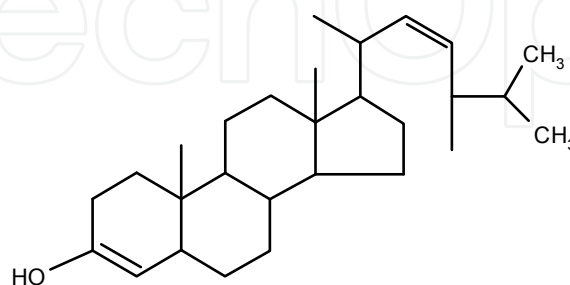


Figure 11. Spinasterol or bessisterol, hitodesterol, 7.22-estigmastidienol. Chemical formula $C_{29}H_{48}O$, MW 412.67 and melting point 168–169°C. [52, 53]

7.1. Sources

The main sources of phytosterols are varied and include fruits such as apple, apricot, banana, cherry, fig, grapefruit, navel oranges, Granada, and strawberry; vegetables such as tomatoes, potatoes, spinach, and carrot; vegetable oils such as avocado, cashew, beaver, brown, coffee, corn, cottonseed, linseed, mustard seed, palm, peanut, pine nut, pumpkin seed, rapeseed flower, rice bran, soybean, sunflower, and wheat germ; grains such as corn, rice bran, sorghum, and wheat; legumes such as beans, peanuts, and soybeans; spices like basil, dill, ginger, white mustard, oregano, paprika, red pepper, poppy seed, and turmeric among others [51].

The hypocholesterolemic effect of phytosterols is attributed to three metabolic actions: inhibition of intestinal cholesterol absorption by competition for the formation of cholesterol mixed micelles; reduction of cholesterol esterification in enterocytes by inhibiting the activity of acyl CoA cholesterol acyl-transferase enzyme, and stimulation of the flow of cholesterol from the intestinal lumen into enterocytes to increase the activity and expression of a conveyor. The joint action of phytosterols on these mechanisms produces a decrease in total plasma cholesterol and LDL cholesterol without changing the levels of HDL cholesterol. Since phytosterols are more lipophilic than cholesterol itself – a property derived from the characteristics of greater length and sidechain complexity – sterols and stanols competitively displace cholesterol from the mixed micelle formed by the action of phospholipids and bile salts in the intestinal lumen. Thus, upon contact with the mixed micelle formed by brush border microvilli on intestinal cells, phytosterols take the place of cholesterol. Cholesterol does not emulsify (moved from the micelle), cannot be absorbed and is eliminated by bowel movements. Phytosterols and phytostanols have low absorption in the intestine, so for the transfer of fatty acids and monoglycerides from the micelle to intestinal cells, which results in the disassembly of the mixed micelle, sterols and stanols would be released along with the unabsorbed cholesterol, finally being excreted in the stool. This would be the first level action of sterols. Intestinal absorption of phytosterols is extremely low (less than 0.5–1.0%) and that of phytostanols is even lower. However, when these sterols are absorbed, inhibition of ACAT (second level action) occurs, whereby cholesterol is not efficiently esterified and incorporated into chylomicrons and stimulates the flow into the intestinal lumen of non-esterified cholesterol. Sterols produce an over-expression of genes encoding proteins of the ABC transporter structure, thus accelerating the flow of cholesterol (third level action) [54].

7.2. Importance

The importance of these natural components is such that several authors state that the inclusion of phytosterols in the diet decreases the concentration of cholesterol in the serum. However, there are very few studies that prove these assertions. In 1999, the Food and Drug Administration (FDA) declared esters of phytosterols as to be Generally Recognized as Safe (GRAS) for use as ingredients in spreads based on vegetable oils in amounts not exceeding 20%, i.e. 20g per 100g of product. [55] In 2004, the Commission of the European Community allowed the

use of phytosterols in food, restricted to consumption to 3 g/d maximum, and these components must be declared in the list of ingredients by percentage and g/100g or g/100ml of product.

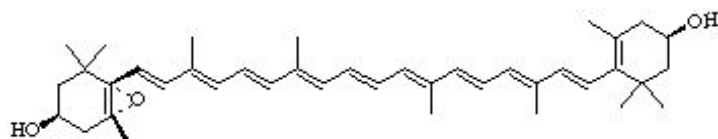
8. Carotenoids

8.1. Carotene

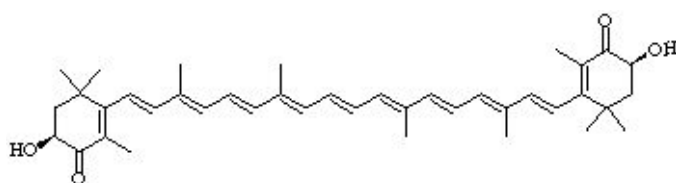
Carotenes are chemical compounds whose absorption spectrum (β -carotene) shows two absorption peaks between 400 nm and 500 nm, corresponding to the colors blue and green, with the reflected red-orange-yellow light gives the compounds their characteristic color. They can reduce the chances of heart attacks, function as soluble antioxidants, and increase the efficiency of the immune system. They have been shown to reduce the likelihood of incidence of some cancers, but may increase the risk of lung cancer in smokers.

According to WHO, dietary factors are involved in about 30% of cancers in Western countries and in 20% of cancers in developing countries. There is ample evidence implicating the involvement of these compounds in such diseases. Lycopene (the pigment that gives tomatoes and watermelons their color) is one that has long been considered as a strong candidate for cancer prevention. The risk of prostate cancer decreases as the consumption of lycopene, α -carotene, β -carotene, β -cryptoxanthina, lutein and zeaxanthin (Figure 12) increases. The consumption of tomatoes, squash, spinach, watermelon and lemons is inversely related to the risk of prostate cancer [57].

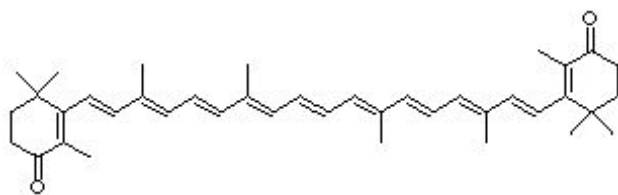
Dark green and orange plants are good sources of β -carotene. Most β -carotene is administered as a synthetic supplement containing a single molecule called all-trans- β -carotene. The β -carotene-contained in foods consist of two molecules: all-trans- β -carotene and 9-cis β -carotene, although the proportion of both varies from one source to another. Initially, no significant difference between natural and synthetic beta carotene was observed, but it is possible that natural beta carotene has differences from the synthetic form. The absorption of carotenoids is affected by digestion of pigments, the presence of hydroxyl groups, and the degree of esterification. Astaxanthin absorption (AXT) occurs mainly in the pyloric caeca, while canthaxanthin (CTX) is absorbed throughout the gastrointestinal tract. AXT is transported mainly by high density lipoproteins (HDL). Vitellogenin is involved in the transport of AXT in liver, muscle and ovaries in mature females. The liver acts as body's main buffer of compounds transported by lipoproteins and consequently a high proportion of absorbed carotenoids are metabolized and excreted in the bile, making them unavailable for muscular pigmentation. When ingested, β -carotene is converted to vitamin A in the mucosa of the small intestine, and is stored mainly in the liver as retinol esters. β -carotene may also be absorbed and stored in fatty tissue without being modified, producing a slightly yellow or orange coloration on the palms of the hands and soles of the feet [57, 58].



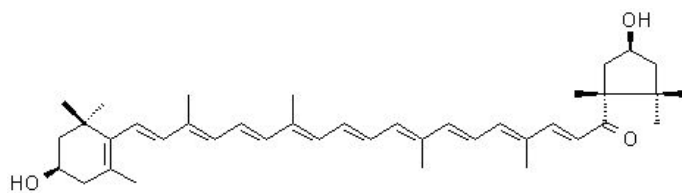
Antheraxanthin: Present in many plants, especially maize



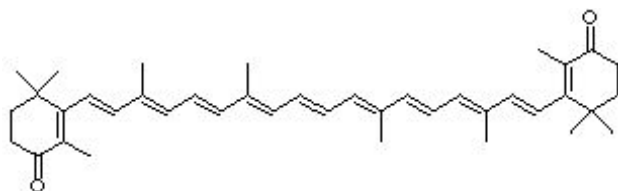
Astaxanthin: Present in salmon, shrimp, lobster, flamingo feathers, and algae



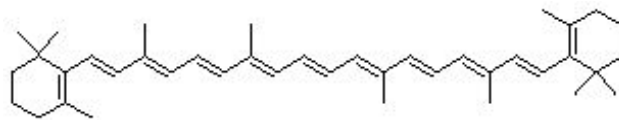
Canthaxanthin: Present in salmon, shrimp, chanterelle and other mushrooms, algae, flamingo feathers



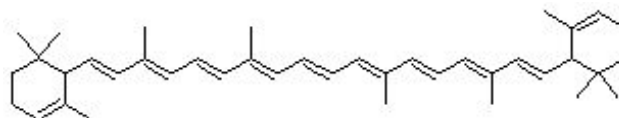
Capsanthin: Present in peppers, paprika



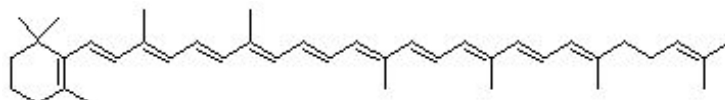
α -carotene: Present in: carrots, most green plants



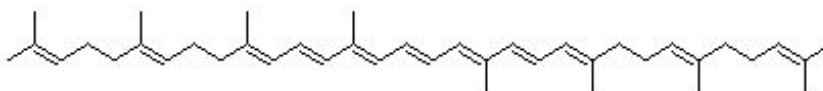
β -carotene: Present in carrots and most other plants



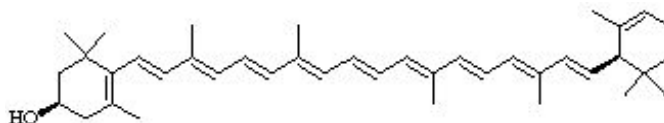
ϵ -carotene: Present in most green plants



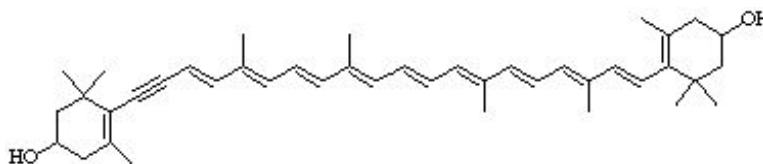
γ -carotene: Present in many plants, often with β -carotene



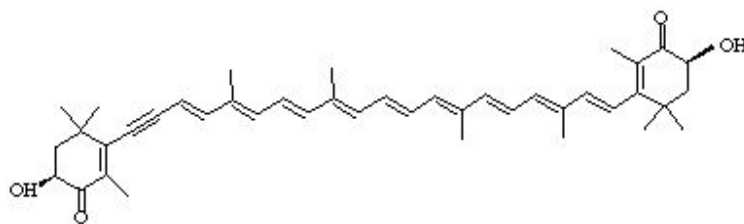
ζ -carotene: Present in many plants



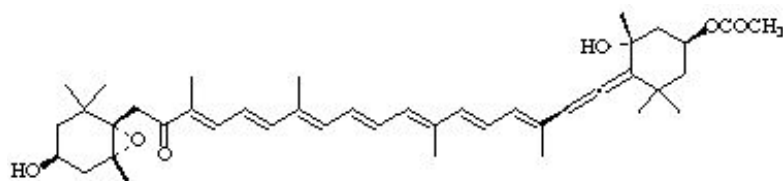
α -cryptoxanthin: Present in many colored plants, including maize and papaya



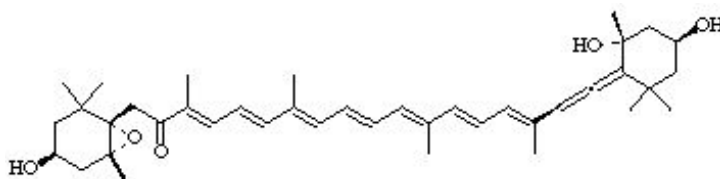
Diatoxanthin: Present in algae and corals



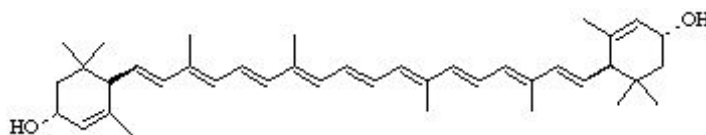
7, 8-didehydroastaxanthin: Present in salmon and crustaceans



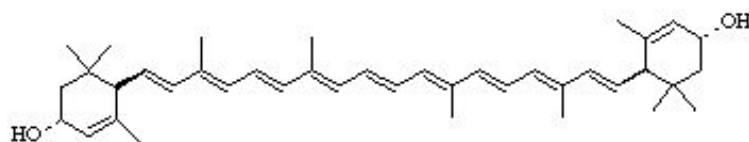
Fucoxanthin



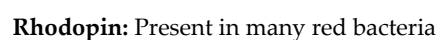
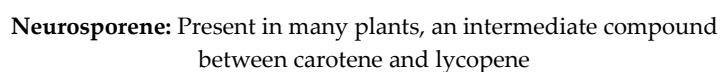
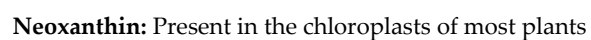
Fucoxanthinol: Present in many algae and seaweed

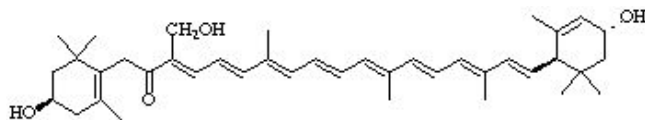


Lactucaxanthin: Present in algae

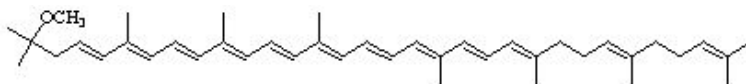


Lutein: Present in many green plants

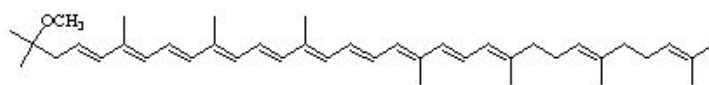




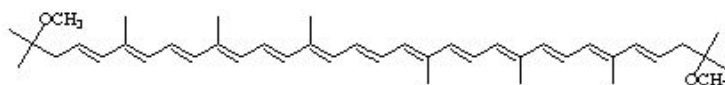
Siphonaxanthin: Present in many red algae



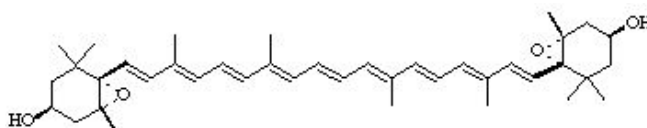
Spheroidene: Present in many red bacteria



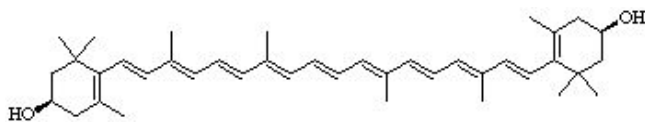
Spheroidenone: Present in many red bacteria



Spirilloxanthin: Present in many red bacteria



Violaxanthin: Present in many plants, especially from the *Viola* family (pansies)



Zeaxanthin: Present in many plants, especially in maize

Figure 12. The most important structure of carotenes [56]

8.2. Lutein and related compounds

Lutein is the most abundant carotenoid in nature, and the main xanthophyll of the protein complexes responsible for uptake of light and pigmentation in plants. It is widely distributed in vegetables, flowers, leaves and fruits, and is involved in the processes of photosynthesis in plants.

8.3. Sources

Lutein is found in grains and dark green vegetables like kale, spinach, broccoli, cabbage, celery, spinach, and asparagus. Corn also contains good amounts as do wheat and fruits like oranges, melon, guava and pear. Table 5 contains the most important approximate values of this component.

Food	Content (mg/service)
Cooked spinach.	20.4
Raw spinach	3.8
Cooked cabbage	14.6
Corn	2.2
Black beans	2.2
Broccoli	1.6
Romaine lettuce	1.3
Green beans	0.8
Egg	0.3
Orange	0.2

Table 4. Lutein content in selected foods

8.4. Importance

Low concentrations of lutein increase the risk of macular degeneration, one of the principle causes of blindness in the elderly. As one of the main macula pigments, lutein is necessary for light absorption and for its antioxidant actions. Light entering the eye can cause photo-oxidation of cells of some eye components, but the pigments overlying the macula offer protection to it. Along with zeaxanthin it filters wavelengths between blue and green in the visible light spectrum. This filtration reduces both the chromatic aberrations produced by this wavelength and the oxidation induced by blue light (similar to damage from UV light). This photo-toxic damage contributes to the development of cataracts and macular degeneration [59].

In Mexico, people over 65 years of age who consuming acetylsalicylic acid daily, have an increased risk of macular degeneration. Usually these are patients are suffering from health problems such as diabetes, obesity and cardiovascular disease. The pattern is further complicated by a high incidence of renal failure and hypertension. Related macular degeneration (ARMD) in these patients is characterized by the progressive loss of central vision to eventual total blindness. Globally, there are currently about 80 million people with blindness caused by ARMD, with 80% of these patients having the wet (or exudative) form and 10–12% have the dry form (geographic atrophy). The main risk factors are age, smoking, race, pale iris, arterial hypertension, cardiovascular disease, hypercholesterolemia, low levels of antioxidants, a diet low in antioxidants, and high exposure to UV light of any kind [60, 61].

9. Minerals or inorganic nutrients

9.1. Role of specific antioxidant minerals

9.1.1. *Selenium*

Selenium is a rare element in the crust of the earth and is almost always found in its native form in metal sulfides. As selenide it is found in association with various minerals such as copper pyrites. In addition to its biological significance to humans and animals, selenium is useful in coloring glass, and in ceramic glazes which give a reddish color. It is an excellent semiconductor and as a result is very useful in the electronics industry.

The chemical nature of selenium, which relates it to sulfur, is biologically important as selenium replaces sulfur in molecules with a biological function such as certain amino acids (seleno-amino acids such as selenomethionine and selenocysteine) and hence selenoproteins form part of the human body (Figure 13). In a similar way to sulfur forming hydrogen sulfide (H_2S), selenium forms Hydrogen selenide (H_2Se) [62].

9.1.1.1. *Metabolism*

It has been observed that there is relationship between the functions of selenium and vitamin E and methionine. When vitamin E interacts with selenium and methionine, a greater effect is achieved against the action of free radicals, both in mitochondria and microsomes. The main metabolic function of selenium is as a prosthetic group of glutathione peroxidase (GPx) and it forms part of its active site. This seleno-enzyme, discovered in 1973, destroys hydrogen peroxide and other oxidantsorganic hydroperoxides, which prevent oxidative damage to cell membranes. By regulating the concentration of glutathione peroxidase and selenium, lipid peroxidation and oxidation of the sulfhydryl groups of the erythrocyte cell membrane — a process that can cause hemolysis — is prevented. Glutathione (Figure 14) is a major component of the red cell membrane such that the balance between the formation of disulfide bridges (GS-SG) and the presence of reduced glutathione (GSH) maintains functionality of the cell membrane [63].

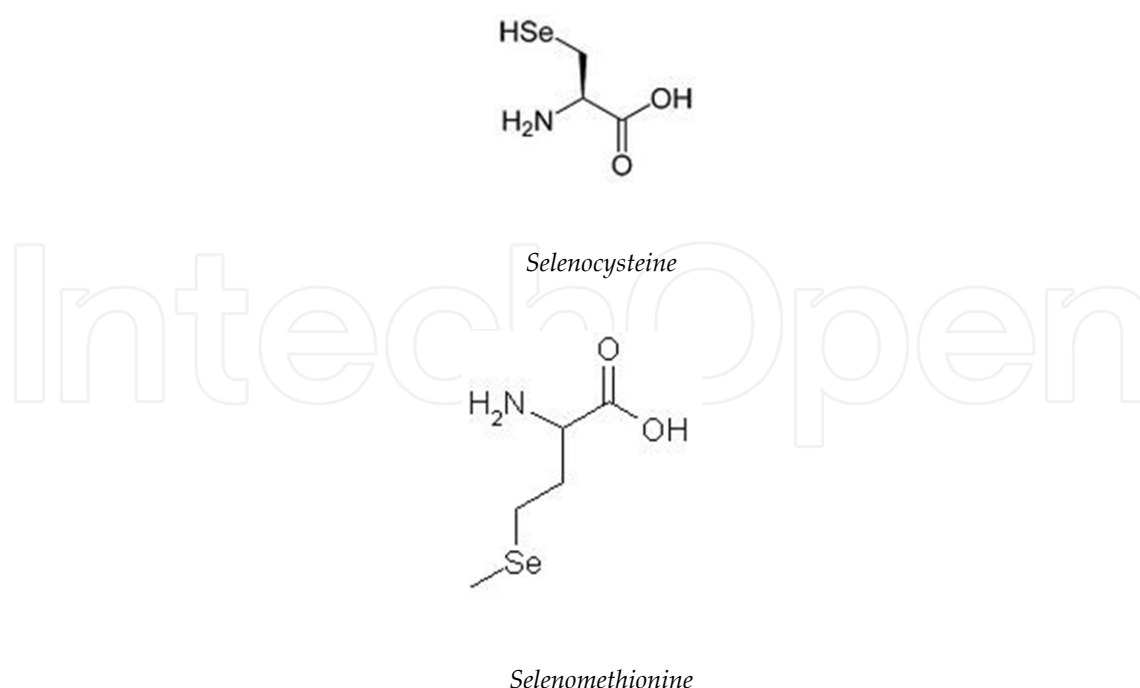


Figure 13. Chemical structure of seleno-amino acids

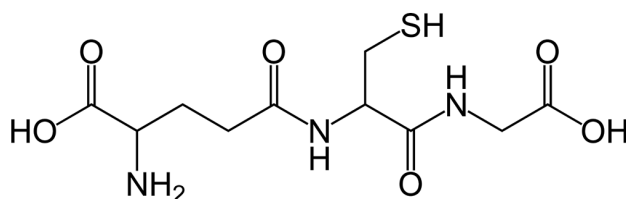


Figure 14. Structure of glutathione (GSH)

The metabolism of selenium in humans is different from that in other animals. The activity of the glutathione peroxidase (GPx) selenium unit is higher in the blood of some laboratory animals in higher primates. For example, blood samples from humans contain approximately 0.1 mcg Se/mL of blood. The GPx/selenium index is very similar in the blood of rats and in that of some species of monkey, but in the blood of rhesus monkeys and humans this index is low. The correlation between blood selenium and GPx activity in the blood of subjects from different areas of the world is significant and linear. This observation comes from studies in China, New Zealand and the United States (Oregon and South Dakota). Selenium accumulates more rapidly in subjects consuming selenomethionine by ingesting selenates, by enabling selenium to form the methionine complex of many proteins, including hemoglobin, more easily. There are two selenoproteins in erythrocytes, while plasma has three: GPx, selenoprotein P, and albumin. Any of these three selenoproteins can serve as a transport protein. In GPx and albumin, selenium is bound to methionine at the active site, and in the case of selenoprotein P, to cysteine [64].

Figure 15 shows the general form of selenium metabolism from its ingestion to excretion. It can be seen that the digestion of selenium in food and water, regardless of the chemical form, starts in the stomach, forming selenite, selenates and free inorganic selenium, and then selenomethionine and selenocysteine are formed through the action of digestive proteases, which are absorbed in the intestine. They are then transported by selenoprotein P to the various tissues and cells that require them.

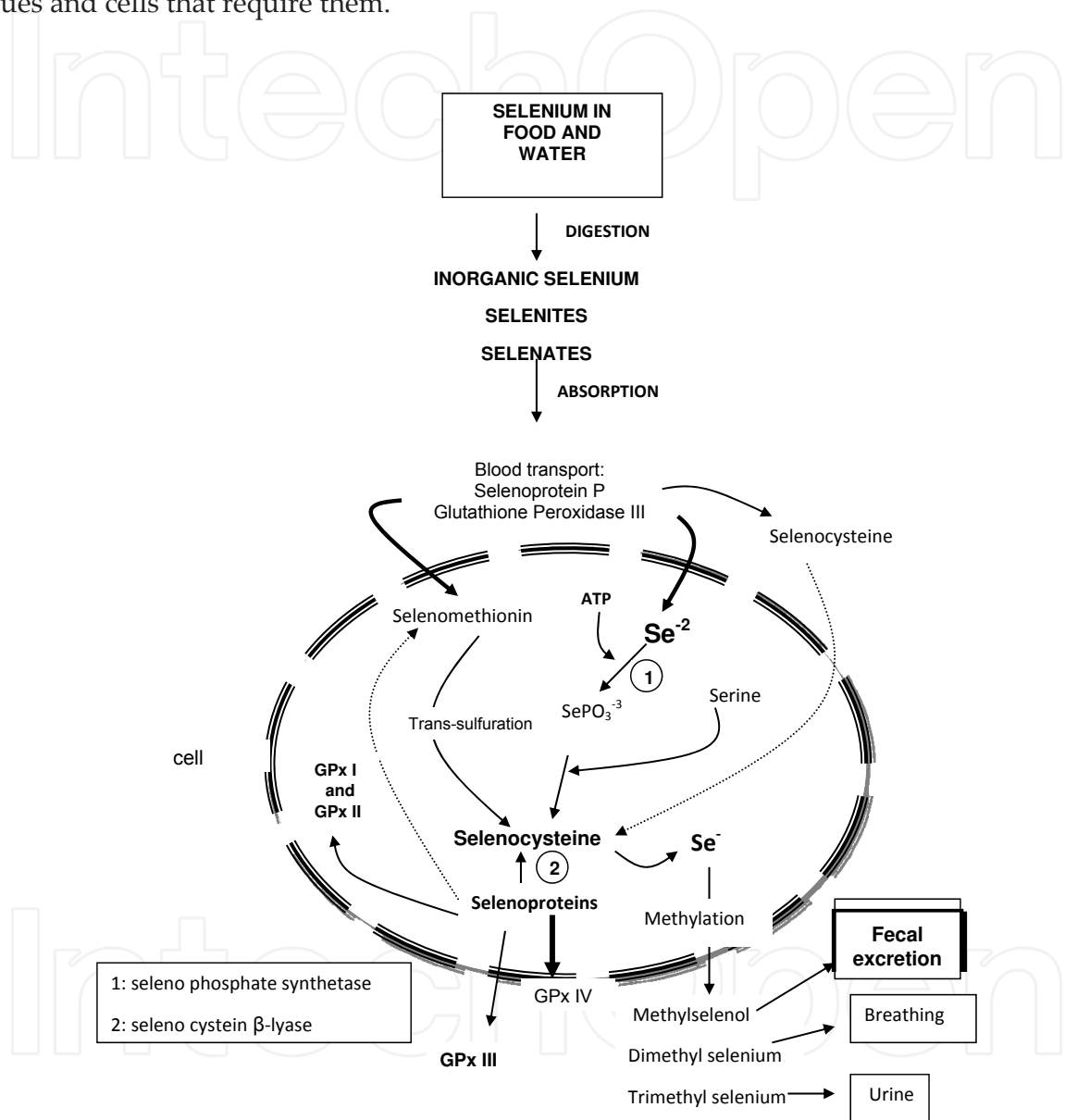


Figure 15. Origin and fate of selenium compounds [62]

Inorganic selenium (Se^{-2}) becomes intracellular phosphate and is incorporated into amino acids (such as selenocysteine). Once incorporated in amino acids, selenium is used in the synthesis of specific selenoproteins such as GPx I and II in the cytoplasm, extracellular GPx III, and membrane bound GPx IV. Unused selenium is methylated and excreted in the urine, feces and breath [63, 64].

9.1.1.2. Requirements and recommendations

The first recommended daily intakes of selenium were made in 1989 in the United States and were 55 and 70 mcg for adult men and women respectively; 40–50 mcg during puberty, 20–30 micrograms for children, and 10–15 mcg per day for infants. Pregnancy increases the recommendation by 10 mcg, and breastfeeding increases it even more. The requirement for selenium is increased with the amount of unsaturated fatty acids (monounsaturated and polyunsaturated) in the diet because of its role in the protection of these fatty acids from free radical attack.

To date, the total extent of selenium requirement has not been fully established. The reference value for the Mexican population is a suggested daily intake (SDI) of between 15–20 mcg in the first year of life of, and 20–55 mcg/d for adult men and women. For pregnant women, an SDI of 60 mcg/d independent of age has been established and for lactating women the SDI is 59 mcg/d. The upper limit of daily intake in adults, without distinguishing between males and females, and pregnant or lactating women, is 400 mcg. For children in the first year of life, an upper limit of 60 mcg is recommended.

9.1.1.3. Source

Some foods that have been identified as important sources of selenium are shown in Tables 6 and 7, but it should be remembered that the concentration of selenium in food depends largely on its availability in the soil and in water used for irrigation. The concentration in the blood of humans depends on the average dietary intake, which varies according to region and tends to follow the pattern of the selenium content of livestock and soil in the region.

Food	mcg/100 g
Brazil nut	Approx 2000
Avocado	11.73
Cauliflower	6.57
Lettuce	6.62
Chili	2.76
Watermelon	9.05
Egg	4.73
Pork	12.37
Beef	8.49
Chicken	8.44
Tuna	10.86
Oyster	5.65
Sardine	44.86
Black beans	3.47
Walnuts	13.53
Cow milk	5.15
Bottled Water	4.56

Table 5. Selenium content of some selected foods [62]

In the case of infants, the ingestion of selenium comes from the mother's milk of. In milk, the selenium content and glutathione peroxidase activity (GPX) is directly dependent on the nutritional status of the mother. After several years of selenium supplementation in Finnish mothers, a significant increase in the amount of selenium in milk and serum was observed. The average content of selenium in human milk is 70 mcg/L from a daily consumption of 85 mcg of selenium. Given a production of 750 mL milk/day, the average daily consumption in children is almost 55 mcg. The effects of supplemental selenium depend on the chemical form in which it is provided to mothers, but is estimated that consumption of 50–70 mcg/day is needed in pregnant women, and that this concentration allows saturation of plasma GPx. The importance of providing selenium in the recommended amounts through breast milk is explained by its antioxidant properties and function in the protection of cell membranes, as these are constantly under "attack" from highly oxidizing free radicals that can destroy them. In an infant, this effect could be devastating and can still affect overall health when pubescent or adult. Selenium deficiency in women can cause health complications from adolescence, along with problems arising from a serious lack of other inorganic nutrients such as iron and zinc that influence reproductive performance [62, 64].

In the case of children who are not breastfeeding, infant formulas with an appropriate selenium concentration are available on the market to meet requirements.

vegetables		fruits		beef		pork		chicken		marine		others	
chard	34,12	blueberries	13,61	res needles	1,1	dried meat	7,38	wing	4,06	cod	8,54	Barbecue	53,66
celery	21,45	Capulín	12,74	Arrachera	67,68	Chicharrón	11,99	heart	2,51	scallops	8,75	snails	28,75
watercress	33,88	Chico zapote	24,91	head	56,13	Chicharrón pressing	7,61	liver	6,18	tent (carpa)	12,42	Chinicuilés °°	0,9
beetroot	20,89	plum	11,56	queue	43,01	chop	9,06	Molleja	2,77	Charales	9,81	quail	18,64
onion	48,59	prune	24,96	heart	62	heart	14,77	drumsticks	11,03	Huachinango	54,42	rabbit	28,48
squash	17,86	Coconut	33,89	Criadilla	2,16	ribs	8,5	breast	9,51	Mojarra	39,33	Escamoles	48,7
Chilaca	36,64	peach	9,6	brisket	0,79	fillet	5,73	neck	7,96	Salmón	17,23	egg quail	12,37
Chilacayote	41,42	strawberry	11,2	fillet	0,91	liver	66,11	rump	2,46	Surimi	17,02	lamb tongue	6,24
Chile	16,17	soursop	10,77	tongue	6,02	sausage	8,6	remnant	7,63	tuna canned in water	35,54	turkey	72,05
cuaresmefio										tuna with vegetables canned	39,28	sausage	
cabbage	24,41	guava	9,53	Milanese	47,71	Milanese °°	5,64	eggs	4,78	Sardine	44,86	sheep brains	5,79
squash blossom	69,04	fig	28,95	ground	0,95	ground	8,66	oils				turkey ham	48,77
beans	82,04	Kiwi	38,08	belly	72,26	Moronga	7,43	canola	20,25	clam	6,61	turkey breast	99,99
Huauzontle	12,82	lychee	10,01	res legs	38,57	pork legs	14,67	cartamo	20,39	squid	23,05	turkey thigh	27,17
Pore	22,9	Lime	19,71	neck	1,04	legs	7,14	canola linseed	24,21	shrimp	74,41	cofee	4,37
Quelite	21,84	Mamey	18,34	Pulp	0,81	kidneys	8,52	corn	15,55	octopus	12,28	sugar	47,9
radish	18,68	blackberry	50,7	Suadero	0,87	brains	10,17	sunflower	11,54	mussel	65,2	salt	2,92
purslane	44,78	pineapple	10,71	gut	68,88	beacon	9,44	walnut	99,25	ostion	5,65	corn tortilla	3,49
Xoconostle	33,91	dominican banana	12,4	liver	7,84	butter	7,78	peanuts	72,25	tuna canned oil	10,86	bread	3,38
mushrooms (setas)	28,95	grapefruit	37	kidney	12,1	ham	4,5	almond	55,04	catsup	3,04	cow milk	5,15
garlic	28,33	Tuna	25,99			loin	12,37	pistachio	68,52	water	4,56	cream	3,89
parsley	56,44	sweet potato	18,19							soda (cola)	5,96	chesse	2,11

Table 6. Selenium content of Mexican foods (mcg Se/100g edible portion) [65, 66]

9.1.1.4. Selenium deficiency

Selenium deficiency is rare in well-nourished populations and can be prevented by administering selenium. The first case of deficiency was reported in the Chinese province of Keshan. The disease occurs frequently in adolescence, with initial symptoms of symmetric stiffness,

swelling and pain in the interphalangeal joints of the fingers, followed by generalized osteoarthritis affecting the elbows, knees and ankles. Selenium deficiency was observed in malnourished patients when feeding over a prolonged parenteral time, and very low selenium concentrations were also observed in the serum of patients fed enterally. Endemic selenium deficiency causes myopathy, but it is unknown whether the deficiency is caused by a particular agent, such as a viral infection, or by some type of metabolic stress [65]. Other seleno-proteins are found in muscle, selenium carrier proteins, xanthine dehydrogenase and some bacterial enzymes. In bacterial systems, selenium-dependent enzymes have been identified such as glycine reductase. In microorganisms, the amino acidic selenium transfer RNA portion is incorporated. Selenium reduces the toxicity of mercury, cadmium and other toxic metals and functions as an important scavenger of toxic metals that are usually ingested in the diet or through the airways. Selenium deficiency prevents purging and thus the toxic action of these metals is unaffected [66].

9.1.2. Zinc

Because of the frequent and widespread consumption of zinc, deficiencies were not observed until 1955, when it was demonstrated in pigs. The appearance of para-keratosis was a sign that humans may be deficient in zinc, and it was noted in malnourished Chinese patients with very low zinc concentrations during World War II. In 1956, zinc deficiency in humans was demonstrated. Since 1961, it has been known that hypogonadism and dwarfism, endemic in rural populations of Iran, are caused by zinc deficiency and this was important in the study of zinc deficiencies in the clinical field and for the implications for public health. In 1963, the role of zinc in human health was unknown. In 1961, zinc deficiency was suspected when teens in Iran suffered from a syndrome resulting in stunted growth, sexual maturation, splenomegaly, and anemia and iron deficiency. Later, in 1963, studies in Egypt established that a zinc deficiency in humans caused a delay in growth and the presence of hypogonadism in men. Since its discovery just four decades ago, a dietary zinc deficiency has been described in children in many countries. Over this period, several researchers have shown that zinc deficiency causes the syndromes mentioned, mainly in children, sometimes coupled with a susceptibility to lower immune protection. Chinese researchers reported a growth failure due to lack of zinc in 30% of children, and 70% of children had low zinc concentrations in their plasma. Analysis of zinc supplementation in nine countries in Latin America and the Caribbean, eight in North America and Europe, five from Asia and the Middle East, and three from Africa was performed, showing a highly significant effect on improving size and body weight in children under 13 years of age. Over the past decade, the therapeutic uses of zinc are mentioned in Wilson's disease, in acute and chronic diarrhea in children, and in the treatment of colds and Alzheimer's disease. Zinc affects multiple aspects of the immune system. It is crucial for normal development and cell mediated immune function. Macrophages are also affected by zinc deficiency, phagocytosis, intracellular killing and cytokine production. Zinc deficiency also affects the functions of B and T cells, through disruption of basic biological functions at the cellular level. Zinc is needed for DNA synthesis and for RNA transcription in cell division and activation. Programmed cell death (apoptosis) is enhanced in the absence of zinc. The function and secretion of cytokines, the basic messengers of the immune system, are

adversely affected by zinc deficiency. Zinc has the ability to function as an antioxidant, preventing cell membranes from being attacked by free radicals, and helping to improve the inflammatory process caused by injuries. Zinc deficiency causes a deterioration in lymphoid tissues and the thymus. It has been observed that immature T cells contain lower amounts of zinc. With a diet deficient in zinc, in a span of two weeks, a deterioration in the ability to prevent or counteract tumor development was observed. Zinc distributed in tissues, contains about 1.4–2.3 g, and 20% of this amount is in the skin, bones and teeth, although most is found in muscles. Only 2% of the total content is in the blood. Higher zinc concentrations are found in the sperm, prostate and epididymis. Based on the body containing around 0.3 mmol Zn/g (20 mg Zn/g), it is estimated that the total body concentration of Zinc in a newborn child is approximately 0.9 mmol (60 mg). During growth and maturation, the concentration of zinc in the human body increases to approximately 0.46 mmol / g (30 mg / g). The total content of zinc in the adult body is about 2.3 mmol (1.5 g) in women, and 3.8 mmol (2.5 g) in men. Zinc is present in the organs, tissues, fluids and secretions of the body. The zinc ion is mainly intracellular with almost 95% of zinc found within cells. Zinc is associated with all the organs but approximately 60–80% of cellular zinc is in the cytosol [67, 68].

9.1.2.1. Function

Zinc participates in the synthesis or degradation of major metabolites such as carbohydrates, proteins, lipids, and nucleic acid reactions. More than 200 enzymes are involved in the stabilization of the structure of proteins and nucleic acid, the integrity of subcellular organelles and transport processes, immune function, and regulation of gene expression, and proteins involved in such regulation are amino acid structures containing zinc. Zinc is also now identified as an antioxidant for the liner to combat the action of free radicals [69]. The metallothionein protein is a non-enzymatic low molecular weight cysteine-rich protein, with a higher zinc content and lower amounts of copper, iron, cadmium and mercury. It has no known biological action, but it acts in metal detoxification or inhibits thionine function, influencing the metabolism of sulfur amino acids. Zinc is abundant in the nucleus, where it stabilizes the structure of ribonucleic acids (RNA) and deoxyribonucleic (DNA) and it is required for the activity of RNA polymerases important in cell division. Zinc also acts on the chromatin proteins related to transcription and replication. Zinc is found in the crystal structure of bones, in bone enzymes and at the zone of demarcation. It is thought to be necessary for proper osteoblast activation, bone forming enzymes such as alkaline phosphatase, and calcification. When minimal bone resorption occurs, there is no zinc found in the bones [70].

At least 70 enzymes contain zinc, 15 are activated by zinc and, more importantly, almost all metabolic pathways require some zinc, e.g., carbonic anhydrase and carboxypeptidase A (pancreatic hexopeptidasa) require one molecule of zinc per mole of protein. The erythrocyte carbonic anhydrase, is 33% Zinc. One of the most important functions of zinc is its role in the synthesis of proteins and nucleic acids, as already mentioned. Zinc deficiency is reflected in the inability to incorporate thymidine into DNA. The ratio of zinc to insulin, and probably glucagon and ACTH, is of great importance even though their role in the function of the hormone is not well established. The correct ratio of zinc to insulin is necessary in order to

achieve its function, along with the presence of other metals such as cadmium and cobalt. Zinc may act at the level of the beta cells of the pancreas in the storage and preparation of hormones. Zinc is involved in the secretion of the adrenal steroids, stimulated by adrenocorticotrophic hormone (ACTH) [71]. Zinc variation in the body may influence the development of infection by affecting the growth of microorganisms or by altering the host response. It is known that the growth of *Aspergillus niger* is dependent on zinc and almost all living systems require zinc to exist. Zinc is involved in various enzymatic systems of bacteria and viruses (aldolases, proteases, polymerases, transcriptases, phosphatases, etc.). When zinc is added to culture media deficient in zinc, it increases the production of DNA, RNA and protein synthesis in bacteria. Zinc favors the stability and production of alkaline phosphatase in *E. coli* in part of the membrane and stabilizes many microorganisms. If there are high concentrations of zinc, then initial rhinovirus growth is inhibited in HeLa cells by inhibition of viral DNA synthesis in BS-1 cells infected with the herpes simplex virus. The alterations that produce infectious diseases in the host are very complex and include metabolic, biochemical and hormonal changes, with iron, zinc and copper involved in these changes. During the acute phase of infection, iron and zinc are moved to the liver, while copper leaves the liver and moves into the plasma, carried by ceruloplasmin. It is rapidly produced during inflammatory and infectious processes and it has been shown that zinc is stored in the liver and is thought to be bound to metallothioneins [72, 73]. Mobilization of plasma zinc to the liver is controlled by LEM (leukocyte endogenous mediator), which is produced in vitro by neutrophils and activated macrophages. This substance has been found in systemic infectious diseases, and has been observed in processes such as cellulite and shigellosis pielonefritis, at low concentrations as in viral infections. Zinc concentrations vary during different stages of infection. For example, during the acute phase, during the effervescence of the disease increases the amount of zinc, and during recovery, zinc concentration normalizes. Zinc is involved in a number of host defense mechanisms helping to maintain the stability of the cell membrane: at increased concentrations, zinc reduces or suppresses phagocytic function, and at low values T cells become dysfunctional. It is involved in the synthesis of nucleic acids and proteins and forms part of various metalloenzymes, like alkaline phosphatase, carbonic anhydrase and carboxypeptidases. Finally, zinc decreases concentrations favoring growth and reproduction of pathogenic germs. Zinc is very important in lymphocyte function. For example, in patients with *acrodermatitis enteropathica*, caused by a chronic deficiency of zinc, defects in cellular immunity result from dysfunctional T cells while B cells function normally. [74, 75]

9.1.2.2. Recommendations and requirements

Zinc requirements depend on the following criteria: the amount required to maintain balance in the body; the amount required to replace endogenous losses; and the amount needed to maintain the normal functions of zinc in the body. With an intake of 12.5 mg of zinc/day through a mixed diet, it should be possible to achieve a proper zinc balance. The RDA provides for at least 15 mg/day of zinc in adolescent and adult males, while in the case of women the amount recommended is 12 mg/day. The recommended amount during the first year of life is 5 mg/day [68, 70].

9.1.2.3. Sources

Food differs extensively in zinc content. The zinc concentration of oysters is 75 mg/100 g, while eggs only contain 0.02 mg/100 g. Chicken meat has 1 mg/100 g, and seafood, beef and other red meats are the best sources of zinc. Cereal grains are relatively rich in zinc; most bran contains zinc in the husk, and about 80% of the zinc is lost when the grains are processed or when milled. There is no uniform enrichment of cereal grains with zinc, although some cereal manufacturers do enrich as standard practice. Nuts and legumes are a relatively good source of zinc. The concentration of zinc in these foods can increase if they are grown in zinc rich soil, or if treated with fertilizers rich in zinc. Intake of zinc depends on the choice of food consumed. Animals, especially meat products, provide about 70% zinc. Cereals are the primary source of zinc. The zinc content of mixed diets varies between 10–15 mg/day and often depends on the amount of dietary protein. Diets based primarily on eggs, milk, poultry, and fish have a lower zinc content than those based on seafood, beef and other red meats. For a better view of the sources of zinc see Table 7, where sources of zinc are displayed in descending order of concentration, while Table 8 shows the sources of zinc in mg/100 g of food. These sources are representative of the most widely consumed foods in Mexico [57]. The variations are similar in vegetarian diets and water is low in zinc. During the first six months of life, zinc intake varies according to the form of milk supplied. Breastfed children ingest 1.9 mg per day, since the first month of age ingest up to 2.7 mg per day, while formula-fed continued children ingest 3.6–4.6 mg per day. Human colostrum contains 20 mg of zinc/L, decreasing rapidly so that after one month milk contains only 3 mg, and after two months, 2 mg/L. Children regularly take in 5–8 mg/day. Adolescent girls have a daily intake of 11 mg, while the daily intake of children between 8–13 years of age is approximately 8–10 mg. Most of the population consumes approximately 7–10 mg/day. Pregnant and lactating women have a similar zinc consumption. These differences are related to different energy intakes and not to the zinc content of diets, even if they are of poor nutritional quality or poorly balanced.

Food, (equivalent, service)	mg
Oysters, eastern, ½ cup	113.0
Pacific oysters, ½ cup	21.0
Wheat germ, toasted ¼ cup	4.7
Ground beef, lean, 90 g	4.6
Beef liver, fried, 90 g	4.6
Turkey dark meat, baked, 90 g	3.8
Instant breakfast, one	3.0
Beef enchilada, one	2.3
Baked beans with pork, ½ cup	1.9
Skimmed ricotta cheese, ½ cup	1.7
Pecans, ¼ cup	1.6
Sesame butter, 1 cup	1.6
Dry peanuts, toasted ¼ cup	1.4

Food, (equivalent, service)	mg
Crab, canned, ¼ cup	1.3
Wild rice, cooked, ½ cup	1.1
Clams, canned ¼ cup	1.1
Lobster, cooked, ½ cup	1.1
Edam, 30 g	1.1
Milk, 2 % fat, 1 cup	1.0
Chicken breast, baked 1	1.0
Nuts, English, ¼ cup	0.8
Bagel, 1	0.6
Gingerbread, 1 piece	.6
Egg, 1	0.6
Baked Salmon, 30 g	0.4

Table 7. Sources of zinc [76]

Food	Content in mg /100 g edible portion
Rapeseed meal	24.2
Lyophilized hen egg	12.5
Ground beans	8.4
Cooked spinach	7.2
Breakfast cereal (Fiber One)	6.7
Barra type bun	6.1
Breakfast cereal (All-Bran)	5.6
Bisteck pulp, bovine	5.4
Pumpkin flowers, cooked	5.3
Beef, shoulder	5.2
Broccoli cooked	5.1
Lettuce, romaine	4.7
Coriander	4.7
Ground beef, fried	4.3
Beef, sirloin steak, boneless roast	4.2
Green beans, cooked	4.2
Huauzontle, cooked	4.2
Lentils, cooked	4.1
Peas, cooked	4.0
Cereal protein	4.0

Table 8. Sources of zinc in Mexican food [77]

9.1.3. Iron

9.1.3.1. Features

Iron is a metal considered by ancient civilizations (such as celestial being) sent by God for use as a metal or a pharmacological therapy or remedy. During Roman times, iron had therapeutic uses in treating diseases such as alopecia, acne, dermatitis, weakness, hemorrhoids, gout, pulmonary diseases, diarrhea, vomiting, edema, fever and cystitis. From the seventeenth century onwards the importance of treating iron chlorosis was known. The first descriptions of the importance of iron were made around 1871 and iron is now known to be one of the most abundant metals. Knowledge of the biological importance of the metal has grown, and interest in the iron deficiency known as anemia continues to this day. Iron is involved in various redox reactions such as hydrogen reduction, and is incorporated into carbohydrates during photosynthesis in the presence of ferredoxins. Aerobic metabolism depends on iron, and iron is an active component of the enzymes in the Krebs cycle [78]. Since 1966, over 30,000 articles have been published on iron, on topics such as biology, medicine and nutrition. Between 1985 and 1991 a total of 9,000 items was published describing iron metabolism, metabolic disorders caused by the deficiency or excess of iron, and iron ratio and nutrition. Despite the abundant information that exists on iron today, there are still many unresolved questions and uncertainties about the functions and metabolism of iron, mostly related to nutrition and immunity. An adult male has 3–5 g of iron in the blood, 30–40% of which is in the form of deposits [79].

9.1.3.2. Function

Iron is involved in the transport of oxygen and carbon dioxide, and functions as an active element in the related process of the cellular respiration of enzymes. The involvement of iron in the function of the immune and cognitive systems has not been fully clarified yet, reinforcing the importance of reducing the incidence of anemia worldwide [80].

Hemoglobin is a part of erythrocytes. The iron-containing protein heme is combined with oxygen in the lungs and carbon dioxide in the tissues. Myoglobin – a hemeprotein – also serves as an oxygen reservoir within the muscles. Oxidative ATP production in the mitochondria includes many enzymes containing heme and non-heme iron. Cytochromes, are involved with respiratory enzymes, several cofactors and the electron transfer chain, for energy production and storage, through oxidation-reduction processes ($\text{Fe}^{2+} \rightarrow \text{Fe}^{3+}$). It is also known that iron is important to the immune system, but the mechanisms by which it acts remain unknown. Neutrophils, white blood cells that engulf and destroy bacteria, are less effective when there is an iron deficiency. Transferrin and lactoferrin protect against infection by preventing microorganisms from using iron for growth. Another feature of iron is the effect it exerts on cognition, especially during childhood, where it improves psychomotor function and learning processes as well as attention and memory, especially in children with anemia. It is important to mention that in adults only 5–15% of iron from food is absorbed, only 2–10% of iron from plants is absorbed, and 10–30% from animal protein. This absorption also depends on several factors such as age, health status, iron status, the condition of the gastrointestinal tract, the amount and chemical form of the iron ingested, and the amount and proportion of various

organic and inorganic components of the diet. Absorption occurs mainly in the duodenum in ferrous form. Iron absorption also depends on the chemical composition in which it is available. Amino acids, such as histidine and lysine, help in the absorption of iron [80, 81].

9.1.3.3. Requirements and recommendations

The requirement for iron and increased hemoglobin depends on the growth rate of the individual concerned. For example, growth is rapid during childhood and adolescence. There are various data that establish the average iron requirement. In children, the amount is 0.35–0.7 mg/day, and from 0.3–0.45 mg/day in pre-menstrual girls. An iron requirement of 38 mg/day for children of both sexes between 4- and 14-years-old, has been suggested.

Other iron requirements are based on the needs of the organism. For example, iron demands are increased in pregnant or menstruating women, and during the first two years of life due to the rapid growth into adolescence. Iron requirements during the first year of life are 0.4–1 mg/day rising to 1.5–2 mg/day during puberty. It is noteworthy that it has been difficult to accurately calculate iron requirements, since each individual varies in their capacity for absorption and the amount of iron available in food.

9.1.3.4. Recommendations for iron [70]

Because iron absorption is very limited, the recommendations are far superior to the requirements due to different situations such as pathological bleeding, and menstruation in the case of women. Adolescent girls who undertake strict diets are generally low in iron, pregnancy, lactation, which incidentally is necessary to supplement for 2 or 3 months before delivery.

9.1.3.5. Sources of iron

There are two types of iron in food: heme iron and non-heme or inorganic iron. Heme iron makes up 5% of iron in the diet and is found only in meat products (including poultry and fish). Non-heme iron is the iron in plant foods, and about 50% of the iron in animal products.

The distinction between the two types of iron is important because the body absorbs heme iron (the iron found in meat) much more efficiently. The main dietary sources of iron include organ meats, meats, legumes, cereals, eggs, seafood and nuts. (See Table 9). It has been found that the best source of iron is the liver, followed by oysters, kidney, heart, lean meat, poultry and fish. Dried beans and vegetables are also among the best sources. Some other iron-rich foods include egg yolk, dried fruits, dark molasses, whole grain breads and fortified wines and cereals. Milk and dairy products also contain a good amount. Iron in fortified cereals, flours and breads contributes significantly to the total intake, with fortified infant cereals being an important source prior to 12 months of age.

It is important to recognize that there are both stimulators and inhibitors to the absorption of iron. For example, besides being a source of heme iron, meat has better intestinal absorption which can be doubled in the presence of vitamin C. Other important inhibitors of iron absorption are tannins (found in tea, coffee and chocolate), phytates and oxalates (found in

legumes, green vegetables and some grains). Table 10 shows further sources of Iron, as well as sources of vitamin C to enhance and improve the absorption of Iron. These data were obtained from tables of the nutritional value of the most widely consumed foods in Mexico.

Iron content (mg /100 g edible portion)	foods
<3	single egg yolk, walnuts, peanuts, sesame, masa tortillas, whole grains, cooked legumes (beans, lentils, chickpeas)
3–6	poblano chili, turkey, dried fruit, beef
6–9	raw legumes (beans, lentils, chickpeas), pinole, Seafood
>9	beef liver, chicken or pork, pumpkin seed, dried chilies, cheese, tuna, moringa

Table 9. Guide to dietary iron sources

Food	Edible portion	Iron (mg)
Excellent		
Moronga / black pudding	50 g	22.5
Pork liver	100 g	17.9
Oysters	100 g	13.4
Beef liver	100 g	6.3
Very good		
Beef	100 g	3.2
Spinach, cooked	½ cup	3.2
Beef kidney	100 g	3.0
Shrimps	100 g	2.4
Red beans	½ cup	2.3
Veal	100 g	2.0
Black beans	½ g	1.8
Pork	100 g	1.6
Fortified cereals	30 g	1.5–4.5
Good		
Artichoke	1 unit	1.6
Nopales	100 g	1.6
White fish	100 g	1.2

Food	Edible portion	Iron (mg)
Chicken	100 g	1.2
Peas (arvejas)	½ cup	1.2
Rice	½ cup	1.1
Avocado	½ unit	1.0
Moderate		
Bread	1 slice	0.9
Raisin	¼ cup	0.8
Tortilla (corn)	1 small	0.7
Eggs	1	0.7
Pasta	½ cup	0.7
Broccoli	½ cup	0.7
Lettuce, dark green	1 cup	0.6
Asparagus	½ cup	0.6
Peanuts	¼ cup	0.6

Table 10. Iron sources and associated portion content [70]

There is a high diversity of Mexican foods that are rich in iron, and while it may be of great importance to know the amount of iron in our food, it is also important to increase the amount of it in the Mexican diet. Simply having a wider variety of foods will help us to improve our general nutritional status. This is why at the end of this paper, we analyze and discuss these sources of iron, as well as the importance of the relationship between iron intake and the need for iron supplementation when there is an iron deficiency, and/or the protective role of this element in the immune system in preventing long-term infections and/or malnutrition, which is also caused by the absence of other specific nutrients in the human diet [80].

The mechanism by which iron causes liver damage has been the subject of many studies. Iron overload produced under experimental conditions causes peroxidation of lipids *in vivo*. Peroxidation occurs at an iron concentration exceeding a specific threshold. Once this threshold is exceeded, the hepatocyte is unable to store iron in a nontoxic form, such as ferritin or hemosiderin, and an increase in iron storage occurs, allowing for the production of free radicals and consequent lipid peroxidation. Furthermore, in experimental models of iron overload, abnormalities of liver organelles (mitochondria, microsomes and lysosomes) are described in association with lipid peroxidation. However, studies have not yet confirmed the connection of these abnormalities the relationship to the development of liver fibrosis and cirrhosis. Research conducted at the beginning of the nineties showed that lipid peroxidation leads to increased transcription of the collagen gene and increased collagen production by human fibroblasts in culture [80, 81].

RAW VEGETABLES		COOKED VEGETABLES		CHILLI (PEPPER)		OTHERS		CEREALS	
coriander	39.92	Spinach	129.98	cascabel	30.60	"axayacatl" worms	48.00	all- bran	36.58
Epazote	36.54	Huauzontle	40.76	chipotle	28.00	pork moronga	36.63	precooked (Nestum)	34.87
Yerbabuena	29.10	Quelite	31.10	Chipotle canned	24.79	roasted coffee	21.40	Raice Krispis	22.23
chard	26.62	Eggplant	28.51	pasilla	24.57	coconut oil	19.60	Crunchy Nut	19.51
parsley	23.94	Romeritos	14.64	mulato	21.36	dry mixed for beverages (Nutrichoco)	15.96	Cereal proteínado	18.60
Coffee	23.00	Bróccoli	13.41	Chile piquin con semilla	15.10	almond capulín seed	15.10	Cerelac	18.31
Verdolaga	20.20	green tomato	12.59	guajillo	18.81	pingüica seeds	14.70	Corn Pops	15.58
mallow (Hidalgo)	19.50	Ejote	12.70	ancho black	13.34	cooked crab (Veracruz)	13.30	bran	14.60
mallow (DF)	19.40	lentils	12.37	pickled jalapeño	14.47	ground beans	12.40	Soy	13.70
jundura plum	18.60	Chicharo	12.25	piquin with seeds	14.28	bean cócona	11.50	soybeans	13.60
bellow	17.22	Chilaca	11.98	guajillo dried cooked	13.18	lentils seeds	11.20	Fiber One wheat bran(all- bran)	13.50 13.10
Chepilín	16.78	artichoke	10.03	mulato dried	12.80	pumkin seeds	11.00	Corn Flakes	12.21
Calabacita (Morelos)	16.40	green bean	9.77	moritta	10.25	sesame seeds	10.68		
Alélón (Guerrero)	15.80			guajillo	10.10	blue mass	10.72	FLOURS OF	
plum (Guerrero)	15.40	BREAD				flour for tamales	10.45	spinach	88.00
Tuna pressed red	13.98	Barra type bun	76.90	ONLY SEEDS OF CHILLI		eelgrass seeds	10.39	potatoes	72.40
Hediondilla quelite	13.80	Enriched bread sliced deffated paste	39.40 19.79	chile chipotle	22.49	corn for pozole	10.27	green bean	18.20
cucumber	13.47			chile mulato	21.85	yellow mass	10.22	black bean	13.50
mushroom "escobeta"	13.20	integral bread	12.43	chile pasilla	17.60	Tortilla of yellow corn black bean cooked (Querétaro)	9.87 9.24	molturada Integral	13.4
Endive	12.50			chile guajillo	15.30	toast (tostiricas)	9.46	carrots	12.40
Chaya dehydrated	12.30			guaje, dried	15.20				
Yurquilla curcuma	12.21			chile morita	15.05	bay bean big	9.40		

The name in parentheses indicates the origin of the food or brand. In some cases the original name of the food is maintained, as there is no adequate translation into the English language.

Table 11. Sources of Iron in Mexican Foods (mg Fe/100 g edible portion) [70, 77]

10. Lipids

10.1. n-3 Fatty acids

10.1.1. Oils, fatty acids and phospholipids

Omega-3 fatty acids, DHA (docosahexaenoic acid), EPA (eicosapentaenoic acid), and polyunsaturated fats, especially the omega-3 type present in fish oil, have a protective effect because they decrease the viscosity of the blood, thus reducing the risk of thrombus formation. Monounsaturated fat, whose main representative is oleic acid present in olive oil, also has a protective effect in raising "good" cholesterol (HDL-c) and preventing the oxidation of "bad" cholesterol (LDL-c) [82]. Fish oils have large amounts of polyunsaturated omega-3 fatty acids (n-3 PUFA), DHA and EPA. The richest sources of these are oily fish (salmon, trout, sardines, and tuna). The high content of DHA and EPA in oily fish is the result of phytoplankton (rich in n-3 PUFAs), which contributes to the adaptation of fish to cold waters. In recent years, the ingestion of omega-3 fatty acids has been associated with a significant reduction in coronary heart disease, cardiac arrhythmias, and acute myocardial infarction (considered the leading cause of sudden cardiac death) due to an increased cardiac output, an almost complete inhibition of platelet aggregation, contribution to the prevention of thrombosis, relaxation of smooth muscle in blood vessels, and lower blood pressure. Polyunsaturated long-chain omega-3 fatty acids have been associated with antiarrhythmic, antithrombotic, antisclerotic, and antihypertensive effects, and prophylaxis of coronary disease [82, 83].

Other important sources include green vegetables like purslane, quelites and broccoli. The capacity of consumption of essential fatty acids such as omega-3 and omega-6 in a proper balance and quantity, helps to stabilize the metabolism of fats in the body and is involved in many other organic processes. As a result, cholesterol metabolism, quantity and transport are normalized, reducing the risk of cardiovascular disease. Omega-3 fatty acids are specifically involved in reducing cholesterol carried in low density lipoproteins (LDL), especially the higher risk, smaller and denser particles of LDL known as "bad" cholesterol, and in facilitating an increase in high-density lipoprotein (HDL) or "good cholesterol" that cleans the arteries instead of damaging them. They also have a role in the normal functioning of the endothelium (on the inside of the body's arteries) within which the lesions of atherosclerosis occur. A good balance in the supply of essential fatty acids and the significant contribution of polyunsaturated and monounsaturated fats delays the onset of atherosclerotic lesions. The intake of polyunsaturated fatty acids corrects delayed or adult onset diabetes, which also reduces, in turn, cardiovascular risk [84].

Many studies suggest that replacement of saturated fatty acids in the diet with n-3 PUFA results in enhanced susceptibility of LDL to oxidation. In humans, susceptibility to LDL oxidation is highly correlated with development of coronary stenosis. Adverse effects of n-3 PUFA in LDL oxidation in vitro do not occur in vivo, and thus may explain the protective effect of n-3 PUFAs against atherosclerosis, as has been demonstrated through reduced uptake by macrophages and a lower electrophoretic mobility of LDL group patients ingesting a daily

supplement of 16 g of fish oil for three months compared to a control group of patients without dietary treatment [85, 86].

10.1.2. Sources of Omega 3

In Mexico, there is a real deficiency of these nutrients as consumption of fish (one of the main sources) and green plants is very low. The estimated per capita consumption is only 5–10g of fish per year, making it very difficult to obtain the minimum required amounts from this source. Green vegetable consumption is limited to only a few vegetables per day. Because these nutrients are part of membrane phospholipids, which are crucial for antioxidant defense against free radicals, the failure to include sufficient n-3 fatty acid promotes the lipoperoxidation observed in the development of slowly progressive or chronic degenerative diseases. When a nutrient is missing, it is likely that others are also deficient, and if within these are other antioxidants, the pattern becomes complicated. The development of chronic and degenerative diseases is likely to be promoted not only by the deficiency of antioxidant nutrients, but also by deficiency of other nutrients "working" with the same function. An imbalance in the intake of antioxidants can lead to free radicals having more of a role in the development of the diseases that cause major public health problems in developing countries. However, the solution will always be prevention through dietary education promoting an increased consumption of foods that are rich in antioxidants from the earliest stages of life, and even before birth [87, 88, 89].

In the table, 12 major sources of n-3 fatty acids are shown. The values of alpha linolenic acid (ALA), eicosapentaenoic (EPA) and docosahexaenoic acid (DHA) in edible plants are presented.

The most important sources of omega-3 fatty acids are fish and vegetables. Fish contains a good amount of alpha-linolenic acid, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Vegetables are an excellent source of alpha-linolenic acid, as a precursor to EPA and DHA, but some other vegetables also contain these fatty acids as shown in Table 12.

a-LINOLENIC / CONCENTRATION mg/100g		EPA / CONCENTRATION mg/100g		DHA / CONCENTRATION mg/100g	
Spinach	36.764	Broccoli	4.987	Romain lettuce	6.240
Watercress	29.828	Chard	4.959	Cauliflower	5.743
Yerbamora	22.521	Lettuce orejona	4.792	Coriander	5.347
Parsley	20.139	Romain lettuce	4.375	Nopal	3.514
Purslane	19.774	Papaloquelite	3.817	Orange pepper	3.259
Italian lettuce	16.943	Coriander	2.848	Huitlacoche	3.183
Lettuce heart	13.303	Serrano pepper (green)	2.458	Yerbabuena	3.171
Beetroot	10.835	Cabbage	2.446	Chayote spineless	2.275

a-LINOLENIC / CONCENTRATION mg/100g		EPA / CONCENTRATION mg/100g		DHA / CONCENTRATION mg/100g	
Serrano pepper (green)	10.822	Parsley	2.144	Serrano pepper (green)	2.227
Broccoli	9.615	Xoconostle	2.009	Red onion	2.041
Artichoke heart	8.815	Epazote	1.764	Mushroom	1.857
Poblano Chili	8.279	Milkweed	1.752	Cabbage	1.696
Coriander	7.554	Habanero pepper	1.680	Tomato	1.338
Lettuce sangría	7.269	Echalot	1.393	Round pumpkin (Creole)	1.262
White onion	6.844	Cebollines	1.316	Lettuce orejona	1.213
Lemon peel	6.242	Red onion	1.299	French lettuce	1.183
Peeled lemon	6.229	Pore	1.280	Papaloquelite	1.175
Red pepper	6.193	Huitlacoche	1.242	Chicory	1.171
Lentil seed	6.096	Green beans	1.086	Epazote	1.083
Chard	5.267	Cucumber with peel	1.027	Asparagus	1.044
Green bean	5.236	Mallow	0.889	Xoconostle	1.034
Red onion	4.941	Green beans	0.875	Radishes	0.894
Round pumpkin (Creole)	4.779	Beetroot	0.859	Green beans	0.798
Nopal	4.562	Nopal	0.822	Papalo	0.679
Chile apple	4.550	Poblano Chili	0.784	Beetroot	0.546
Papalo	4.382	Bean sprouts	0.780	Tomato ball	0.538
Chayote boneless	4.234	Round pumpkin (Creole)	0.759	Chayote boneless	0.496
Eggplant	3.761	Chayote boneless	0.699	Artichoke	0.482
Romeritos	3.545	Peeled cucumber	0.691	Swede	0.463
Celery	3.385	Cuaresmeño pepper (jalapeño)	0.673	Parsley	0.450
Cucumber with peel	3.293	Elongated gourd (Italian)	0.657	Lemon peel	0.450
Carrot	3.249	Tomato	0.615	Mushrooms	0.442
Cebollines	3.099	Swede	0.584	Habanero pepper	0.414
Yerbabuena	3.084	Carrot	0.572	Red cabbage	0.355
Cauliflower	3.006	Yerbabuena	0.554	White onion	0.332
Red cabbage	2.974	Xoconostle heart	0.549	Broccoli	0.329
Garlic	2.878	Orange pepper	0.548	Poblano Chili	0.316
Mushroom	2.391	Squash blossom	0.507	Yerbamora	0.312
Chilacayote	2.239	Mushroom	0.497	Cherry tomato	0.248

a-LINOLENIC / CONCENTRATION mg/100g		EPA / CONCENTRATION mg/100g		DHA / CONCENTRATION mg/100g	
Jicama	2.189	White onion	0.469	Huazontle	0.243
Tomato ball	2.039	Red pepper	0.459	Cebollines	0.203
French lettuce	1.976	Cob	0.453	Peas	0.196
Peeled cucumber	1.971	Chile apple	0.354	Cob	0.168
Cob	1.813	Papalo	0.344	Chilacayote	0.150
Orange pepper	1.658	Chicory	0.322	Echalot	0.137
Hongo escobetilla	1.607	Watercress	0.281	Hongo escobetilla	0.075
Pore	1.590	Asparagus	0.263	Eggplant	0.069
Habanero pepper	1.577	Chayote spineless	0.253		
Hongo horn sheep	1.483	Peas	0.241		
Asparagus	1.393	Huazontle	0.239		
Chicory	1.269	Red cabbage	0.229		
Swede	1.265	Tomato	0.135		
Romain lettuce	1.114	Green pepper	0.129		
Milkweed	1.114	Hongo yemita	0.122		
Lettuce orejona	1.093	Radishes	0.118		
Epazote	1.045	Chilacayote	0.067		
Spring onion	1.036	Green tomato	0.047		

Table 12. Most important sources of omega 3 fatty acids in edible vegetables [94, 95]

In Mexico has over 12, 000 km of shoreline and as such is rich in different species of marine fish, and it also has a wide variety of freshwater fish. In the Gulf of Mexico alone, about 500 different species of fish rich in omega-3 have been identified along with many others in the Pacific Ocean, only some of which have been specifically identified and their nutrient composition with respect to omega 3 fatty acids reported. Our group has already published some of these results in international journals and forums [90, 91].

However, the per capita consumption of fish has diminished in recent years, and domestic production has been used mostly for export purposes. Current per capita consumption is approximately 10 g of fish annually, compared with countries such as Japan and China that have a per capita consumption of 70 kg annually. On the other hand, their consumption has been avoided by some groups due to the danger of allergic reactions, or in the case of pregnant women and children under one-year-old, on the recommendation of doctor. In addition, fish oils rich in omega 3 have been used in the preparation of food for pets and animals, rather than for humans [92, 93].

The consumption of green vegetables has also declined, and vegetables are only consumed daily in certain sectors of society, while in others they are essential not consumed at all — as discussed in the text.

There are currently few studies in Mexico showing deficiencies of omega 3 fatty acids, but the presence of several clinical signs of deficiency have already been observed in children. These include prematurity, malnutrition, retardation of intrauterine growth, recurrence of infections and allergies, psychomotor and mental neurodevelopment problems, delays in learning and damage to the vision of young children. In adults, there is an increase in retinopathy and a significant increase in neurodegenerative problems like Alzheimer's disease and other diseases associated with the central nervous system, at younger ages than in other countries.

Among the most serious issues is a significant increase in mortality from coronary heart disease, which is the primary cause of death in Mexico. It should be remembered that omega 3 fatty acids are considered to be cardio-protective. In pregnant women, both eclampsia and pre-eclampsia have been rising at the same time as premature rupturing of membranes and other problems associated with pregnancy. Our group has already documented a very serious deficiency of omega 3 fatty acids in the breast milk of clinically healthy women, and the same deficiency was observed in the children they fed. Even the serious problem of childhood obesity in Mexico is being analyzed from the clinical point of view of micronutrient deficiency, while that of the ingest excesses in others. Thus, increased consumption of foods with a high glycemic index and saturated fat content is combined with the consumption of very few foods with omega-3 fatty acids such as fish and plants. In addition, the high rate of physical inactivity and sedentary lifestyles, makes the problems somewhat more challenging to solve. If we add to this pattern the impending imbalance between consumption of antioxidant and pro-oxidant food, the result will be further disease and mortality — the main problems affecting public health in Mexico [94].

11. Antioxidant supplements and Mexican sportsmen

The consumption of vitamins and inorganic elements via supplements began back in 1930 following the chemical isolation of these compounds. In 1939, cyclists in the Tour de France reported an increase in performance level after taking vitamins. However, in 1940, further research did not significantly support this position (Applegate and Grivetti, 1997), and even in the current literature ergogenic capabilities of these micronutrients are not described. Among the vitamins and inorganic elements frequently used in sport are vitamin B12, antioxidants such as vitamin C, E, beta carotene, zinc, magnesium, copper, selenium and iron, carnitine, chromium, vanadium, and boron.

The significant increase in the demand for oxygen that physical activity involves — especially if it is intense and continuous — is responsible for a parallel rise in the formation of free radicals derived from oxygen (up to three times the resting value) and is considered to be one of the main initiators or amplifying mechanisms of muscle damage associated with exercise (Margaritis, 2003; Alonso, 2006). These highly reactive particles can activate a series of chain

reactions and are able to damage collagen fibers, cell membranes and nuclear structures. The use of antioxidants in sport can be a useful weapon against oxidative stress, since it has been observed that athletes have better levels of vitamin C and glutathione peroxidase, and therefore higher levels of lipid peroxidation.

On this basis, it is easy to understand the importance of mechanisms enabling antioxidants to protect the body from the damage caused by exercise, especially when it is intense. Although it is noteworthy that the unjustified use of vitamins and inorganic elements as a special requirement for better physical performance was observed. Several studies have demonstrated that the use of these supplements can exceed daily recommended amounts in the blood by 10–15 times without causing any direct benefit in athletic performance [96].

In the study by Guzman in 2009 (Table 13 and 14) [97] the ingestion of nutrients by Mexican athletes in different sports was investigated. These sports were weightlifting, tae kwon do, swimming, zumba and spinning. In the same table, the average intake of vitamins and minerals calculated from records of the daily supplements taken by type of exercise, and in brackets the average percentage exceeding the daily recommendation, are shown. Not included in this section is a calculation of the intake of vitamins and minerals from the diet. We found that eating supplements alone met and even exceeded the recommendations for the daily intake of most vitamins in all types of exercise.

Minerals	Weightlifting	Tae Kwon	Swimming	Zumba	Spinning
A ($\mu\text{g/d}$)	1030.5 \pm 1140.5 (132% \pm 169.4)	–	2500 (625%)	3300 (471%)	3333 (476%)
D ($\mu\text{g/d}$)	10 (200%)	–	10 \pm 7.1 (200% \pm 141.4)	17 (340%)	5 (100%)
E (mg/d)	27.4 \pm 21.2 (182% \pm 141.5)	25.83 (172%)	3.3 (47%)	162.1 \pm 127.4 (1080.7 \pm 849.4)	99.9 (666%)
K (mg/d)	28.3 \pm 10.4 (29% \pm 3.2)	–	–	–	–
C (mg/d)	43.1 \pm 61.1 (55% \pm 82.6)	600 (800%)	410 \pm 445.3 (1667% \pm 1747.4)	1000 (1333%)	250 (333%)
B ₁ (mg/d)	8.4 \pm 23.4 (752% \pm 2132.7)	–	1 (167%)	76 (6909%)	50 (4545%)
B ₂ (mg/d)	9.5 \pm 24.6 (848% \pm 2239.9)	–	1.2 (192%)	76 (6909%)	50 (4545%)
B ₆ (mg/d)	9.9 \pm 24.5 (758% \pm 18.8)	–	2 (333%)	72 (5538%)	50 (3846%)
B ₁₂ ($\mu\text{g/d}$)	14.1 \pm 25 (589% \pm 1043.6)	25 (1041%)	–	6 (250%)	75 (3125%)
Niacin	21.7 \pm 24.5	–	6.7	82	50

Minerals	Weightlifting	Tae Kwon	Swimming	Zumba	Spinning
(mg/d)	(144%±174)		(83%)	(586%)	(357%)
Folic	168±105	1000	–	400	200
(µg/d)	(42%±26.2)	(250%)	–	(100%)	(50%)
Pantotenic	17.8±24.7	–	–	76	50
(µg/d)	(356%±494.6)	–	–	(1520%)	(1000%)
Biotina	83.1±55	–	–	300	50
(µg/d)	(284%±183.3)	–	–	(1000%)	(167%)

() Data brackets indicate percentage of the recommended amount

Table 13. Average daily intake and % RDI of vitamins from food supplements (Guzman, 2009) [97]

Minerals	Weightlifting	Tae Kwon	Swimming	Zumba	Spinning
Calcium	391.6±228.8	–	43.4	2000	25
(mg/d)	(39%±23.2)	–	(5%)	(200%)	(2%)
Iron	5.8±6.1	115	–	–	5
(mg/d)	(60%±61.2)	(1045%)	–	–	(28%)
Phosphorus	421.7 ±339.5	–	66.7	228	–
(mg/d)	(58%±49.6)	–	(13%)	(32%)	–
Magnesium	114.5±75.9	–	–	133	25
(mg/d)	(29%±18.8)	–	–	(41%)	(8%)
Zinc	6.5±3.9	–	–	30	7.5
(mg/d)	(66%±49.7)	–	–	(375%)	(94%)
Boron	–	–	–	–	500
(µg/d)	–	–	–	–	(50,000%)
Potasium	99	–	–	–	–
(mg/d)	(5%)	–	–	–	–
Chrome	56.7±34.8	–	–	6	50
(µg/d)	(164%±96.4)	–	–	(24%)	(200%)
Selenium	28.5±16.9	–	–	50	75
(µg/d)	(52%±30.8)	–	–	(91%)	(136%)
Cupper	650±382.1	–	–	600	1000
(µg/d)	(72%±42.6)	–	–	(67%)	(111%)

() Data brackets indicate percentage of the recommended amount

Table 14. Daily average and % RDI of inorganic elements from ingestion of food supplements [97]

12. Antioxidant capacity of foods and their use as nutraceuticals or functional foods

Nutraceuticals are natural biological substances extracted from natural sources that are characterized by their anti-denaturing biotechnological processes, which conserve all original properties without any chemical manipulation. Once extracted from the natural source, nutraceuticals are studied using processes similar to those used to identify the biological properties of drugs in animals and humans. Once their properties are documented, they can be used as food supplements for human consumption, not as substitutes for a normal diet. Sometimes, some of these natural substances may act as potential drugs due to their biological properties and can be prescribed as therapeutic adjuvants for preventive and / or curative purposes. As a concept, the nutraceutical is between a natural raw commodity and a xenobiotic chemical or a substance foreign to the body [98].

In most cases their functional properties are implicit to their use, and for this reason biological testing prior to use is not necessary. The concept of functional foods should not be confused with health foods, which often contain the same components. There are also combinations of essential nutrient with herbs and exotic fruits and and vegetables, somehow giving them any "extra" functional property which makes them very striking [99].

13. Nutraceutical as a therapeutic option

The novelty of these functional foods is the current scientific knowledge of the beneficial properties linked to disease prevention that they possess. Nutraceuticals have been associated with the prevention and treatment of at least four of the diseases that contribute to the high rate of mortality in developed countries (cancer, diabetes, cardiovascular disease and hypertension) and to the prevention of other diseases such as neural tube defects, osteoporosis and arthritis [100].

13.1. Global use of nutraceuticals

The growing expectation for good health has led to a large number of studies demonstrating the effective use of nutraceutical components. Although their indiscriminate use has been speculated, it is reversing the paradoxes of health in each country, which are shown as major public health problems, indicating a serious food deficiency or the mishandling of individual diets. Thus, it is as mentioned as serious public health problems, and in some cases even endemic in the population, diseases like cancer, diabetes mellitus, cardio and cerebrovascular disease, hypertension, obesity, cirrhosis and malnutrition. The concept of prevention is closely associated with the word nutraceutical, and this is a current that is gaining strength as prevention of disease is better than trying to find a cure. The costs to budgets of the latter impacts on virtually every hospital in the world [85, 86].

13.2. Convincing results

The cellular effects of nutraceuticals are easily understood as they are part of the phospholipid cell membrane. Their function is to protect the cells from virtually everything that can damage them such as natural, self or foreign oxidants which can cause lipid peroxidation.

Virtually all of these nutraceuticals act to protect against oxidants commonly called free radicals (FR), making them natural antioxidants (vitamins E, C, Selenium, CLA, etc). These antioxidants are found within — or are associated with — the lipid bilayer of the cell membrane, and many of them are even associated with membrane phospholipids.

Antioxidant protection is based on the union of the nutraceutical with FR, which is different with each nutraceutical, in that some take a shorter or longer time to join to FR, and it is in that space of time where the effectiveness of the nutraceutical reflects its protective biopotency. This may partly explain the anticarcinogenic property shown along with an antioxidant capacity. Most of these results have been obtained by in vivo and in vitro studies and have been confirmed by cell culture. Even where inhibition of carcinogenicity by nutraceuticals has been demonstrated (in conjugated linoleic acid (CLA) for example), it is in part accomplished by inducing the activity of antioxidant enzymes (such as SOD, catalase, GPx) and the generation of cytotoxic activities in certain tumor lines. When an antioxidant (an enzyme, vitamin or mineral) has failed to maintain protection of the cell membrane by itself, the combined activity of several nutraceuticals can achieve the expected protection [102, 103].

14. Conclusions

The use of healthy foods is increasing and is becoming more evidence-based with the use of functional foods and plants —or components thereof — with a nutritional content that can help prevent or treat disease. The conscious and optimal use of these nutraceuticals as antioxidants requires more knowledge of their properties and their experimentation probed epidemiologically. Regardless, higher consumption of vegetables and fruits that contain a lot of these antioxidant free radical scavengers is always recommended. It doesn't matter if the food is raw or cooked, it is more important that daily consumption increases. There is a discussion to be had regarding organic vegetables, and even organic animal products, but it is more important to include them as 50% of the diet, regardless of whether the source is organic or not. The appropriate consumption of fiber, prebiotics and probiotics is also necessary for adequate intestinal health and body processes.

It is controversial, but for energy and other substrates, cells need to oxidize nutrients such as glucose, amino acids or fatty acids. However, diet provides not only the body of nutritious elements, a high amount of consumed compounds have also an important oxidative capacity, and other compounds which are not oxidants per se, can create, once absorbed, an oxidative environment.

The most important advice for people below the poverty line is to recommend to them the high consumption of vegetables, which in Mexico are cheap and easily available, but there are

still issues with directions for their use and handling, and further problems of proper living conditions and, of course, income. Both appropriate nutritional guidance directed at this group to address risk, and high-impact informative programs that influence diet, are lacking. A lack of knowledge of the nutritional composition of food is another factor which is not yet being addressed due to the lack of information and analysis of the same. However, efforts aimed at solving the problems are taking place, but very slowly.

Promotion of the national strategy for better health through the prevention of disease makes use of the available evidence. It includes universal, targeted and specific actions to reduce the effect of CNCD. All actions can only be conducted with the consensus of society and in conjunction with government agencies. In particular, attention should be focused on schools through programs directed at primary and secondary education, and the training of teachers and parents as well as regulation of the food sold on campuses and surrounding areas. The same actions can be applied to institutions that distribute food to their employees. These measures are complemented by demand for the development or adaptation of facilities enabling physical activity.

Agreements with the food industry and any organization related to the distribution of food are necessary, so that the population is allowed access to healthy food, or at least access to information of its composition. In this sense, measures corresponding to the composition of industrialized food labelling are already being taken according to Mexican standard (NOM-051-SCFISSA1-2010), where the nutritional values of the same should be limited, but specific information on industrialized or natural foods is still very limited and restricted to only certain groups of professionals. Information on healthy lifestyles must be disseminated by various means of communication in order to promote regular exercise and the correct choice of foods. Information about the risks of obesity and diabetes alone is not enough to change habits. The effect that this information has will be magnified if an environment conducive to putting it into practice can be created.

Author details

José Luis Silencio Barrita^{1*}, Sara Montañó Benavides² and Santiago Sánchez³

*Address all correspondence to: silenciobarrita@live.com.mx

1 Instituto Nacional de Ciencias Medicas y Nutrición “Salvador Zubirán”, Departamento de Ciencia y Tecnología de los Alimentos, México

2 Instituto Nacional de Ciencias Medicas y Nutrición “Salvador Zubirán”, Departamento de Nutrición Animal, México

3 Instituto Mexicano del Seguro Social, IMSS, Hospital General de Zona No. 30, “Iztacalco”, Departamento de Nutrición y Dietética, México

References

- [1] Clasificación Estadística Internacional de Enfermedades y Problemas Relacionados con la Salud (CIE), INEGI, 2013
- [2] Rivera Dommarco, Juan, et al. Encuesta Nacional de Nutrición 1999. Estado nutricional de niños y mujeres en México. Cuernavaca, México, Instituto Nacional de Salud Pública, 2001. Available in www.insp.mx/enn
- [3] Martínez Jasso I, Villezca Becerra PA. La alimentación en México: un estudio a partir de la Encuesta Nacional de Ingresos y Gastos de los Hogares. *Revista de información y análisis*, 2003; 21:26-37
- [4] Silencio Barrita JL. Ácidos grasos poliinsaturados y selenio en leche humana. *Nutrición Clínica*, 2004; 7(4):227-39
- [5] Córdova-Villalobos JA, Barriguete-Meléndez JA, Lara-Esqueda A, Barquera S, Rosas-Peralta M, Hernández-Ávila M, De León-May ME, Aguilar-Salinas CA. Chronic non-communicable diseases in Mexico: epidemiologic synopsis and integral prevention. *Salud Pública Mex* 2008; 50: 419-427.
- [6] Aguilar-Salinas CA, Mehta R, Rojas R, Gomez-Perez FJ, Olaiz G, Rull JA. Management of the metabolic syndrome as a strategy for preventing the macrovascular complications of type 2 diabetes: controversial issues. *Curr Diab Rev* 2005; 1: 145-158.
- [7] Gutiérrez-Delgado C, Reynoso-Noverón N, Hernández-Ávila M, MoharBetancourt A. Perfil epidemiológico de los cánceres en población adulta mexicana, periodo 2000-2010 [documento en revisión].
- [8] Editorial. El cáncer en México: propuestas para su control. *Salud púb Méx*, 2014; 56(5): 418-420
- [9] Aguilar-Salinas CA, Olaiz G, Valles V, Rios JM, Gomez-Perez FJ, et al. High prevalence of low HDL cholesterol concentrations and mixed hyperlipidemia in a Mexican nation wide survey. *J Lip Res* 2001; 42:1298- 1307
- [10] Aguilar-Salinas CA, Rojas R, Gomez-Perez FJ, Valles V, Rios-Torres JM, Franco A, et al. High prevalence of the metabolic syndrome in Mexico. *Arch Med Res* 2004; 35:76-81.
- [11] Aguilar-Salinas CA, Rojas R, Gomez-Perez FJ, Garcia E, Valles V, RíosTorres JM, et al. Early onset type 2 diabetes in a Mexican, populationbased, nation-wide survey. *Am J Med* 2002; 113:569-574
- [12] Estadísticas a propósito del... día mundial contra el cáncer (4 de febrero)" datos nacionales, 2015 disponible en: [file:// EAP_del_dia_mundial_contra_el_cancer.pdf](file://EAP_del_dia_mundial_contra_el_cancer.pdf)
- [13] http://www.coneval.gob.mx/rw/resource/coneval/info_public/PDF_PUBLICACIONES/Evolucion_Historica_050411.pdf

- [14] Olaiz G, Rojas R, Barquera S, Shamah T, Aguilar-Salinas C, Cravioto P, et al. Encuesta Nacional de Salud 2000. La salud de los adultos. Cuernavaca, Morelos: Instituto Nacional de Salud Pública, 2003.
- [15] Oláiz-Fernández G, Rivera-Dommarco J, Shamah-Levy T, Rojas R, Villalpando-Hernández S, Hernández-Ávila M, et al. Encuesta Nacional de Salud y Nutrición 2006. Cuernavaca, Morelos: Instituto Nacional de Salud Pública, 2006.
- [16] Bourges H. Glosario de términos nutriólogicos. Cuadernos de Nutrición. Vol. 24. No. 1. Enero- Febrero 2001.
- [17] Silencio Barrita JL. Vitaminas: conceptos generales, Nutrición Clínica 2006; 9(3):36-44
- [18] Bourges Rodríguez H. Glosario de términos para la orientación alimentaria. Cuad Nutr 1988; 11(6):1-47.
- [19] Ascencio Peralta C, Bourges Rodriguez H, Gomez Rodriguez FE, Silencio Barrita JL. "Conceptos basicos sobre la nutricion y los nutrimentos ", Fisiologia, Celulas, Organos y Sistemas, editores E. Julio Muñoz Martinez y Xaviera Garcia Gonzalez, Fondo de Cultura Economica, Tomo IV, Capitulo VII.1, Pag 23-45, 1997.
- [20] Braunwald E., Isselbacher KJ. Petersdorf RG., Wilson JD., Martin JB., Fauci AS. Harrison's Principles of Internal Medicine, Mc Graw Hill Co, 11a Ed, 1987, USA.
- [21] Briggs MH. Vitamins in human Biology and Medicine. CRC press, 2nd edition 1987, USA.
- [22] Hoffman-La Roche F. Compendio de vitaminas, 1970 Basilea, Suiza.
- [23] Casanueva E, Valdés-Ramos R, Pfeffer F, Ricalde-Moreno A, García-Villegas E, Meza C. Serum retinol in urban Mexican women during the perinatal period. Salud Pública Mex 1999; 41:317-321.
- [24] Mayor Oxilia R. Estrés Oxidativo y Sistema de Defensa Antioxidante. Rev. Inst. Med. Trop. 2010; 5(2):23-29
- [25] Stephensen ChB. Vitamin A, infection, and immune function. Ann Rev of Nutr. 2001, 21: 167-192
- [26] Huiming Y, Chaomin W, Meng M. Vitamin A for treating measles in children (Review), 2005, The Cochrane Collaboration. Published by John Wiley & Sons, Ltd
- [27] Valkoa M, Leibfritzb D, Moncol J, Croninc MTD, Mazura M, Telserd J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem & Cell Biol, (2007) 39: 44-84
- [28] Geil P, Shane-McWhorter L Dietary supplements in the management of diabetes: potential risks and benefits.
- [29] Doldo E, Costanza G, Agostinelli S, Tarquini C, Ferlosio A, Arcuri G, Passeri D, Scioli MG, Orlandi A. Vitamin A, Cancer Treatment and Prevention: The New Role of Cel-

lular Retinol Binding Proteins. *Biomed Res Int* (2014), Article ID 624627, downloads.hindawi.com/journals/bmri/.../624627.pdf

- [30] Bennisir H, Sridhar S, Abdel-Razek TT. Vitamin A... from physiology to disease prevention. *Int J Pharmaceutical Sci Rev and Res* (2010) 1(1):68-73
- [31] Marks J. A guide to the vitamins, their role in health and disease. MTP Medical and Technical Pub. 1975, England.
- [32] Brigelius-flohe R, Traber MG. Vitamin E: function and metabolism. (1999), *FASEB J*, 13:1145-1155
- [33] Drevon CA. Absorption, Transport and Metabolism of Vitamin E. 1991, 14(4):229-246
- [34] Gagné A, Wei SQ, Fraser WD, Julien P. Absorption, Transport, and Bioavailability of Vitamin E and its role in Pregnant Women. *J Obstet Gynaecol Can* 2008; 31(3):210–217
- [35] Wu JH, Croft KD. Vitamin E metabolism. *Mol Aspects Med* 28 (2007) 437–452
- [36] Dutta A, Dutta SK. Vitamin E and its Role in the Prevention of Atherosclerosis and Carcinogenesis: A Review. *J Am College of Nutr*, (2003); 22 (4):258–268
- [37] González J, Pérez N, García G. Vitamina E: niveles séricos en hombres, mujeres y embarazadas. *Bol Med Hosp Infant Mex*. 1982; 39: 327-332.
- [38] Vega-Franco L, Meza C, Meijenrik J, Alegret C. Concentración de vitamina E en niños con desnutrición proteico-energética. *Bol Med Hosp Infant Mex*. 1989; 46: 607-610.
- [39] Garcia-Closas R, Berenguer A, Tormo MJ, Sánchez MJ, Quiros JR, Navarro C, Arnaud R, Dorronsoro M, Chirlaque MD, Barricarte A, Ardanaz E, Amiano P, Martinez C, Agudo A, Gonzalez CA. Dietary sources of vitamin C, vitamin E and specific carotenoids in Spain. *British J Nutr* (2004), 91, 1005–1011
- [40] Silencio Barrita JL.- Antioxidantes en el tratamiento de las enfermedades: Vitamina C. Capítulo 28.- En el libro: Los antioxidantes y las enfermedades crónico-degenerativas, tomo II, editores Morales González JA, Fernández Sánchez AM, Bautista Ávila M, Vargas Mendoza N, Madrigal Santillán EO. Universidad Autónoma del Estado de Hidalgo, 2009.
- [41] Iqbal K, Khan A Ali Khan Khattak MM. Biological Significance of Ascorbic Acid (Vitamin C) in Human Health: A Review *Pakistan J Nutr* 2004, 3(1):5-13,
- [42] Simon J.A Vitamin C and cardiovascular disease: a review *J Ame Collage of Nutr*, 1992, 11 (2):107-125
- [43] Simon JA, Hudes ES, Tice JA. Relation of serum ascorbic acid to mortality among US adults. *J Am Coll Nutr* 2001; 20:255–63.

- [44] Bourges H, Madrigal H, Chavéz A., Tablas del valor nutritivo de los alimentos mexicanos. Publicación L-12 INN, 1983, 13 ed. México
- [45] Harris J.L. Vitamin C saturation test. Standardization measurements at graded levels of intake. *Lancet* 1943, 1:515,
- [46] Head KA, Ascorbic Acid in the Prevention and Treatment of Cancer. *Altern Med Rev* 1998; 3(3):174-186)
- [47] Jacob RA, Pianalto FS, Agee RE. Cellular ascorbate depletion in healthy men. *J Nutr.* 1992; 122:1111-1118.
- [48] Gomez E, Silencio JL, Bourges H. Vitamin B2, B6 and C status in patients with primary Sjögren's syndrome. 7th International congress of mucosal immunology, august 16-20, 1992, Prague Csechoslovaquia.
- [49] Silencio Barrita JL, Santiago Sánchez MS. CHAPTER 18.-Antioxidant role of ascorbic acid and protective effects on chronic diseases. In the book *Oxidative stress and chronic degenerative diseases- a role for antioxidants*, edited By JA Morales-Gonzales, In Tech Editors, sept 2012, pages 449-484. ISBN 980-953-307-650-3
- [50] Herrera, V. Más evidencia en contra del uso de vitaminas y antioxidantes en la prevención de enfermedades crónicas. *Evidencia Actualización en la Práctica Ambulatoria-2002*, 5(6): Nov-Dic
- [51] Watt B.K. A.L. Composition of foods. Agriculture Handbook num. 9, United States, Departament of Agriculture, USA, 1975.
- [52] The Merck index a n enciclopedia of chemicals and drugs, eighth edition published by Merck &CO., Inc Rahway NJ USA, 1968
- [53] Moreau RA, Whitaker BD, Hicks KB. Phytosterol, phytostanol and their conjugates in foods: structural diversity, quantitative analysis, and health promoting uses. *Progress in Lipid Research.* 2002; 41:457
- [54] Duperon R. Thiersault M. Duperon P. *Phytochemistry*, 1984; 23:743
- [55] Hicks KB, Moreau RA. Phytosterols and phytostanols; functional food cholesterol busters. *Food Technology*, 2001; 55(1):63
- [56] <http://dcb-carot.unibe.ch/carotint.htm> and <http://www.carotenoidsociety.org/>
- [57] Sahyoun, R.N., 1996. Carotenoids, vitamins C and E and mortality in an elderly population. *Am. J. epidemio*, 144: 501-511
- [58] Kumar S, Pandey AK, Rao MM, Razzaque WAA. Role of β carotene/vitamin A in animal reproduction. *Veterinary World*, 2010; 3(5):236-237
- [59] Andrews S, 2001. JANA, the role of lutein in human health. *J Am Nutraceutical Assoc* 4(2):1-30

- [60] Gonzalez Cortes JH. Desgarro de epitelio pigmentario en la degeneración macular relacionada con la edad. XXV Congreso Mexicano de Oftalmología, Morelia Mich, 3-7 agosto 2002, disponible: <http://dmremonterrey.blogspot.mx/>
- [61] Clinicopathological correlation of retinal pigment epithelial tears in exudative age related macular degeneration: pretear, tear, and scarred tear. *Br J Ophthalmol*. 2001 April; 85(4): 454-460
- [62] Silencio Barrita JL. El selenio. *Cuadr Nutr* 2011, 34(6):230-235.
- [63] Silencio Barrita JL, Bellido Villa A, Ritter Santiago T. Selenio. *Nutrición Clínica* 2004, 7(1):78-85
- [64] Silencio Barrita JL. Las enfermedades renales y el selenio, *Nutrición Clínica* 2003, 6:291-292
- [65] Ramiro Anaya M. Determinación de selenio por un método fluorométrico en 100 alimentos seleccionados. Tesis experimental, Facultad de Ciencias, UNAM, 2005.
- [66] Rivero Silva OM. Determinación de selenio en 200 alimentos mexicanos. Tesis experimental, Instituto Nacional de Ciencias de la Salud, Universidad Autónoma del Estado de Hidalgo, 2010.
- [67] Casanueva E, Kauffer Horwitz M, Pérez Lizaur AB, Arroyo P. *Nutriología Médica*, Editorial Panamericana, México, 2ª edición, capítulo III, 2001.
- [68] Ortiz-Andrellucchi A. Nutrición e inmunidad. *Rev Soc Med Quir Hosp Emerg Pérez de León* 2007; 38(Suppl 1):12-18.
- [69] Hunt J, Murphy M, Martner P, Faraji B, Swendseid M, Reynolds R, Sanchez A, Mejia A. Zinc, vitamin B6, and other nutrients in pregnant women attending prenatal clinics in Mexico. *Am J Clin Nutr*. 1987; 46: 563-9
- [70] Bourges RH, Casanueva E, Rosado JL. Recomendaciones de ingestión de nutrimentos para la población mexicana: bases fisiológicas, tomo 1, Editorial Médica Panamericana, México, capítulos V, VI y VIII, 2005.
- [71] Osredkar J, Sustar N. Copper and Zinc, Biological Role and Significance of Copper/Zinc Imbalance. *J Clinic Toxicol* 2011, S3:001
- [72] Naithani M, Bharadwaj J, Darbari A. Review of Various Indicators for Assessment of Zinc Requirement and Effectiveness. *Acta Medica International* 2014; 1 (1):32-35
- [73] Hambidge M (2000) Human zinc deficiency. *Journal of Nutrition* 130: 1344S-1349S
- [74] Hambidge M (2003) Biomarkers of trace mineral intake and status. *Journal of Nutrition* 133: 948S-955S
- [75] Nriagu J. Zinc Deficiency in Human Health in http://www.extranet.elsevier.com/homepage_about/mrwd/nvrn/Zinc%20Deficiency%20in%20Humans.pdf

- [76] Raymond JL. Krause's Food, Nutrition and Diet Therapy, 13th ed., W.B. Saunders Co, 2010
- [77] Hernández M, Chávez A, Bourges H. Valor nutritivo de los alimentos mexicanos: tablas de uso práctico. México: Instituto Nacional de la Nutrición, División de Nutrición, 1977
- [78] Sarika Arora and Raj Kumar Kapoor (2012). Iron Metabolism in Humans: An Overview, Iron Metabolism, Dr. Sarika Arora (Ed.), ISBN: 978-953-51-0605-0 Intech on line Books.
- [79] Lash A, Saleem A. Iron metabolism and its regulation. A review. *Ann Clin Lab Sci*, 1995; 25(1): 20-30
- [80] Carocho M, Ferreira ICFR. A review on antioxidants, prooxidants and related controversy: Natural and synthetic compounds, screening and analysis methodologies and future perspectives. *Food and Chem Toxicol*, 2013; 51:15–25
- [81] Brambilla D, Mancuso C, Scuderi MR, Bosco P, Cantarella G, Lempereur L, Di Benedetto G, Pezzino S, Bernardini R. The role of antioxidant supplement in immune system, neoplastic, and neurodegenerative disorders: a point of view for an assessment of the risk/benefit profile. *Nutr J*. 2008, 7:29
- [82] Simopoulos AP. Human requirement for N-3 polyunsaturated fatty acids. *Poult Sci*. 2000 Jul; 79(7):961-70.
- [83] Silencio Barrita JL, Guzmán Federle D, Molano Pérez F. implicación de los ácidos grasos n-3 en nutrición y diabetes, en el libro *Nutrición y Diabetes*, editores Solano Solano G, Del Castillo Arreola A, García Meras M, Guzmán Saldaña R, Romero Palencia A, compilado por la Universidad Autónoma del Estado de Hidalgo, 2011, páginas 283-316, ISBN 978-607-482-192-5
- [84] Silencio Barrita JL. LCPUFAS y su importancia en la alimentación del recién nacido. *diccionario de Especialidades Farmaceuticas en Pediatría*. PLM, ed Mauricio Aguilera. MEXICO, 2010, ISBN 978-607-7767-20-6
- [85] De Vriese SR, Dhont M, Christophe AB. Oxidative stability of low density lipoproteins and vitamin E levels increase in maternal blood during normal pregnancy. *Lipids* 2001; 36(4):3616.
- [86] Agostoni C. The difficult balance between dietary polyunsaturated fatty acids. *Acta Paediatr* 2003; 92(12):13713
- [87] Lands WEM. Biochemistry and physiology of n-3 fatty acids. *FASEB J* 1992; 6:2530-6.
- [88] Ramírez-Silva et al.: Fatty acids intake in the Mexican population. Results of the National Nutrition Survey 2006. *Nutrition & Metabolism*, 2011 8:33.
- [89] Silencio Barrita JL, Lara Flores G, Pérez Gil Romo F, Montaña Benavides S, Ortiz Huidobro RI, Castro González MI, Barrera Millán E, de Titto Carboni AM, López

- Cabrera FT, Santiago Sánchez MS, Falcón A, Irisson R. Ácidos grasos en el calostro y en la leche madura de mujeres mexicanas, 2012, *Rev Mex Ped*, 79(1):5-12
- [90] Silencio Barrita JL, Ortiz Ortega V., Castro González MA, Montaña S, Velasco S, Cárdenas B, Pérez-Gil Romo F., Bourges Rodríguez H. Trace element content of marine foods from the pacific mexican coast. *J Trace Elem Exp Med* 1998; 11(4): 471
- [91] Castro González MI, Silencio Barrita JL, Juárez Silva ME, Montaña Benavides S, Pérez-GIL Romo F. Composición química proximal de la fauna de acompañamiento del camarón de Veracruz. (Golfo de Mexico). *Biologia Tropical* 46(2):249-256, 1998
- [92] Castro González MI, Pérez Gil Romo F, Carranco Jáuregui ME, Montaña Benavides S, Silencio Barrita JL. Vitaminas y minerales de sardina en salsa de tomate, colectada en las zonas pesqueras del pacifico mexicano. *Arch Latin Nutr* 1999, 49(No. 4): 379-383
- [93] Castro González MI, Ojeda A, Silencio Barrita JL, Cassis L, Ledesma H, Pérez-Gil F. Perfil lipídico de 25 pescados marinos mexicanos con especial énfasis en sus Ácidos grasos n-3 como componentes nutraceuticos, *Archivos Latinoamericanos de Nutrición*, 54 (3):328- 336, 2004
- [94] Castro-González I, Maafs-Rodriguez AG, Silencio-Barrita JL, Galindo-Gómez C, Pérez-Gil F. Evaluation of the possible inclusion of certain fish species in chronic kidney disease diets based on their adverse and beneficial nutrient ratios. *Int J Food Sci Nutr.*, 2012, 63 (4): 1-7
- [95] Martínez Pérez D. Contenido de ácidos grasos en verduras producidas y consumidas en México, tesis licenciatura en nutrición Universidad SIGLO XXI –UAEM, febrero 2014, México.
- [96] Williams MH. Dietary supplements and sports performance: introduction and vitamins. *J Int Soc Sports Nutr.* 2004; 1(2):1-6.
- [97] Guzmán-Federle D. Evaluación del consumo de complementos alimenticios en individuos que realizan ejercicio físico en gimnasios y centros deportivos de Pachuca, Hidalgo, tesina 2009, Universidad Intercontinental, México
- [98] Silencio Barrita JL. Investigación clínica con respecto a Nutrición y cáncer. *Rev Mex Ped* 2009; 76 (No. 4), jul-ago:181-186.
- [99] Biruete Guzman A, Juarez Hernandez E, Sieiro Ortega E, Romero Viruelas R. Silencio Barrita JL. Los Nutraceuticos. Lo que es conveniente saber. *Rev Mex Ped* 2009; 76(3): 136-145.
- [100] Kalra EK. Nutraceutical- definition and introduction. *AAPS Pharm Sci* 2003; 5:1-2
- [101] Nutraceuticos. En: http://www.maimonides.edu/gerontologia2007/2007/03/las_dianas_que_se_utilizan_con.html.
- [102] Silencio J.L. Nutraceuticos, *Nutri informate* 2006, 11(4): 11-12

- [103] Hawker N. Nutracéuticos, ¿alimentos o medicamentos? Junio 2003. www.vitafoods.co.uk/2002/nutrition/ffnut.htm.

IntechOpen

IntechOpen