Antioxidant Capacity of Food

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ABSTRACT

Reactive oxygen species (ROS) are usually produced by the living cell and have different functions in its normal activity. However, if they are produced excessively, or if the system that keeps them in controlled levels is deficient or insufficient, a phenomenon known as oxidative stress is produced. This is related to the aging process and the onset and development of several diseases as well as their complications such as endothelial dysfunction in cardiovascular disease concomitant with the oxidation of low density lipoproteins and aggravated by smoking, the appearance of advanced glycosylation end products in diabetes mellitus. In neurodegenerative disorders such as Alzheimer's disease, neuron plasma membrane malfunction is caused by phospholipids peroxidation, leading to cell death.Cancer develops from genetic mutations resulting from DNA damage.Taking this into account, several antioxidant properties have been studied in different foods, beverages and spices, including their relationship with the prevention of degenerative diseases. Antioxidants obtained through diet can act in different ways: first, preventing the excess of free radicals, thus avoiding oxidative damage to the cell. Secondly, after damage has occurred, antioxidants can control free radical levels preventing further damage thereby alleviating some symptoms caused by oxidative stress. In this review, some basic concepts of reactive oxygen species, oxidative stress and antioxidants are analyzed, as well as some of the direct and indirect methods used to assess the antioxidant capacity of foods. Some of the food highly consumed by the Mexican population has antioxidant capacity and it is herein summarized.

Key words: Antioxidants, Food, Reactive oxygen species, Oxidative stress, Methods to assess the antioxidant capacity, Mexican food.

INTRODUCTION

Reactive oxygen species

Molecular oxygen is relatively harmless and it is essential for living cells in order to obtain energy. However, some reactive species are derived from oxygen during aerobic metabolism (as a sub product of mitochondrial respiration), or from environmental conditions (pollution, smoke, radiation, drugs). These are termed reactive oxygen species (ROS) and they exhibit two unpaired electrons in different orbitals at their highest energy level, which makes them susceptible to the formation of radicals.[1,2]

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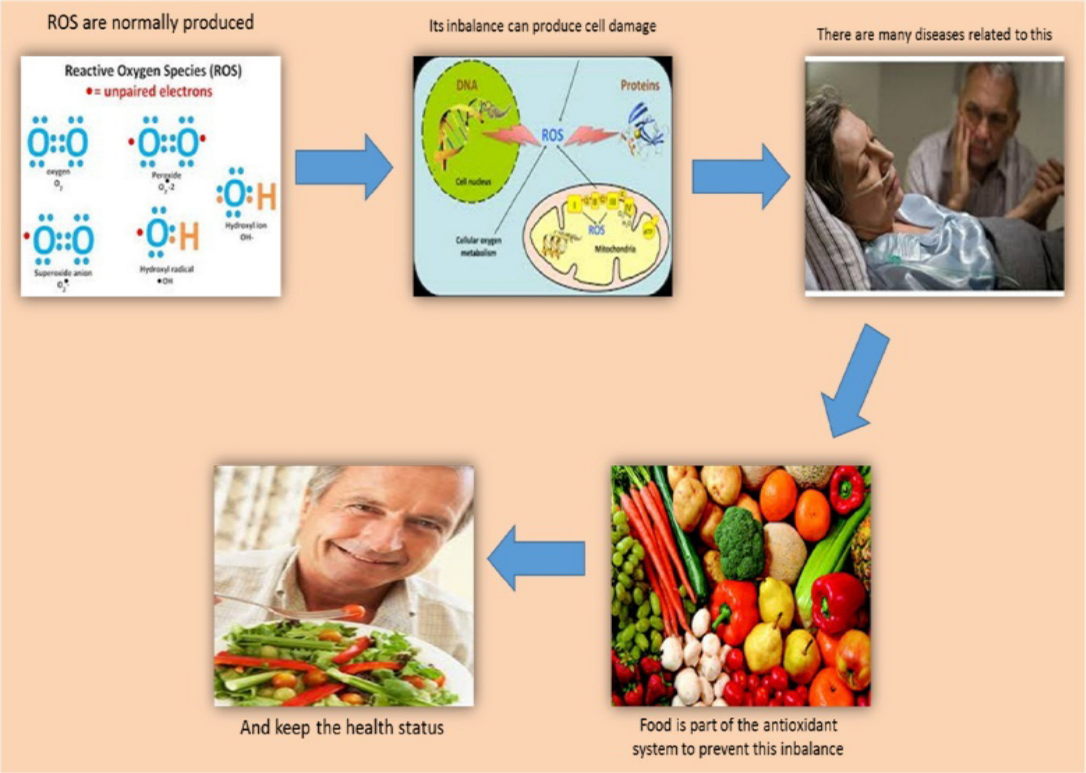
DOI: 10.5530/fra.2016.1.1

ROS rapidly and indiscriminately react with virtually all types of biomolecules such as nucleic acids, free nucleotides, proteins, lipids and carbohydrates, causing damage, mutations, protein inactivation and cell death. However, when present in low to moderate levels, both ROS and reactive nitrogen species (RNS) show beneficial effects in several cell responses and immune functions. Therefore there has to be a delicate balance between their production and elimination within the organism.[1,2]

ROS is a collective term that describes free radicals derived from oxygen, such as superoxide anion ($O2^{\bullet}$), hydroxyl radical ($HO^{\bullet}$), peroxyl radical ($RO2^{\bullet}$) and alkoxy radical ($RO^{\bullet}$), as well as hydrogen peroxide, a non-radical species resulting from oxygen metabolism (H_2O_2).[3]

Mitochondria are the main source of ROS. About



Graphical Abstract

1-2% of the oxygen consumed by this organelle results in ROS production, mainly through the activity of the electron transport chain. Some of the responsible enzymes of this process are: Ubiquinol: cytochrome c oxidoreductase (Complex III), NADH: ubiquinone oxidoreductase (Complex I) along with succinate:ubiquinone oxidoreductase (Complex II), and the flavoproteins: Acyl-CoA dehydrogenase, glycerol phosphate dehaydrogenase, monoamine oxidase, dihydroorotate dehydrogenase, pyruvate and α-ketoglutarate dehydrogenase.[3,4]

Oxidative Stress

Oxidative stress is the result of increased exposure to oxidative agents or the decline in protection against them, or both simultaneously. Oxidative stress is the cause of a wide spectrum of genetic, metabolic and cellular responses, such as necrosis, which modulates gene expression, apoptotic response, among others.[5]

Oxidative stress has been also implicated in the etiology of several diseases (>100) as well as the aging process. The most investigated diseases are: endothelial dysfunction in cardiovascular disease concomitant with the oxidation of low density lipoproteins (LDL) and aggravated by smoking, the appearance of advanced glycosylation end products in diabetes mellitus, the presence of stress, etc. In neurodegenerative disorders such as Alzheimer's disease, neuron plasma membrane malfunction is caused by phospholipids peroxidation, leading to cell death. Cancer develops from genetic mutations resulting from DNA damage. Free radicals have also been linked Diabetes mellitus and its complications, such as persistent hyperglycemia leading to oxidative stress and a)glucose autoxidation, b)non-enzymatic glycosylation, and c) polyol pathway that further produces oxidants.[6,7]

How to measure oxidative damage

There are direct and indirect methods to assessthe impairments caused by oxidative stress:

Direct

- Concentration of oxidative agent. This is a complicated method because of thevery short half-life of the oxidative species (e. g. the hydroxyl radical has a half-life of 10^{-10} seconds). Electron spin resonance spectroscopy is the only analytical technique that directly detects ROS, although it is not applied to humans.
- ROS detection in blood. Transition metals, once free from their chelated form (a form that is usually present in plasma and cells), they possess the

Table 1: Antioxidant protection system

Antioxidant	Type
Endogenous	Bilirubin
	Thiols: Glutathione, lipoic acid, N-Acetyl cysteine
	NADPH y NADH
	Ubiquinone (Coenzyme Q)
	Uricacid
	Enzymes:
	Copper/zinc and manganese dependent superoxide dismutase
	Iron dependent catalase
	Selenium dependent glutathione peroxidase
Diet	Vitamin C
	Vitamin E
	Beta-carotene, Carotenoids and oxicarotenoids: Lycopene and lutein
	Polyphenols: Flavonoids, flavones, flavonols, proanthocyanidins, among others,
Metal binding Proteins	Polyphenols:
	Albumin (Copper)
	Ceruloplasmin (Copper)
	Metallothionein (Copper)
	Ferritin (Iron)
	Myoglobin (Iron)
	Transferin (Iron)

Taken from: Antioxidants.[11]

ability to catalyze redox reactions.The products from such reactions are scavenged by phenolic compounds resulting in coloured solutions that can be characterized by spectrophotometry.

Indirect

- Quantification of oxidation end products. ROS are indirectly measured through the assessment the of oxidized products derived from proteins (which accumulate carbonyl groups after they are attacked by ROS), DNA (it produces over 12 metabolites when reacting with ROS), and lipids (their peroxidation is evaluated by quantifying compounds reacting with thiobarbituric acid, aldehyde detection, volatile hydrocarbures in exhaled air and fluorescent compounds resulting from lipid peroxidation).
- Antioxidants as biomarkers. Several studies show that antioxidant levels can increase or decrease with different diseases, so they can be used as markers for therapeutic purposes. Antioxidant enzymes (superoxide dismutase and catalase), non-enzymatic antioxidants (vitamins C, A, E and ubiquinone), and DNA repair enzymes (redox endonucleases) are all modulated according to oxidative stress levels.
- Immunohistochemical markers used in toxicological

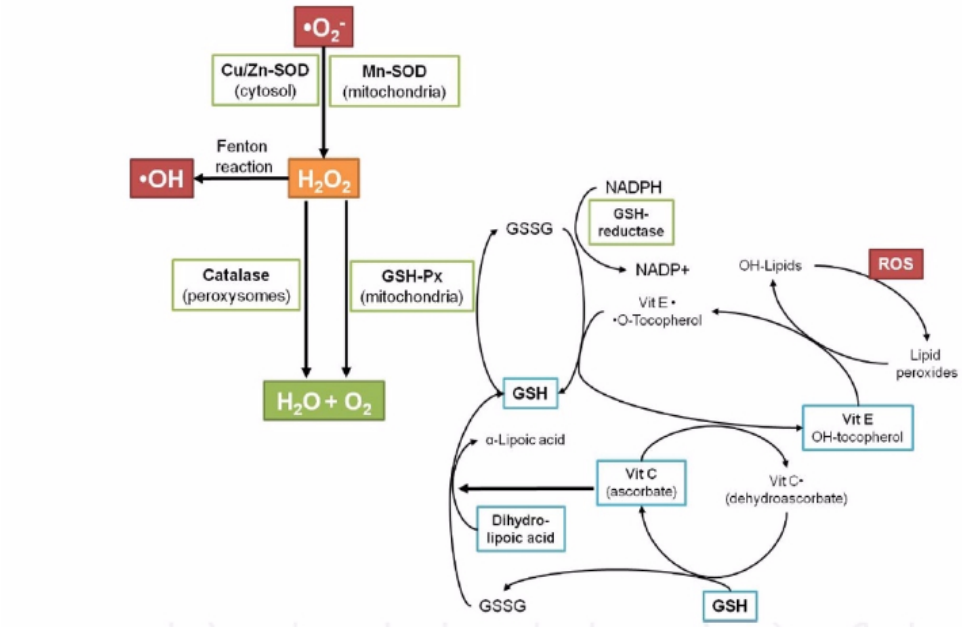


Figure 1: Antioxidant mechanisms (enzymatic and non-enzymatic)
Small amounts of the oxygen consumed by aerobic metabolism will be converted to superoxide anion. This species must be scavenged or converted into less reactive (and less harmful) molecules. The main enzymes that regulate this process are Superoxide dismutase (SOD), Glutathione Peroxidase (GSH-Px) and Catalase. There are also nonenzymatic antioxidant mechanisms, which contribute to convert GSSG back to GSH. Antioxidant vitamins such as A, C, E and alpha-lipoic acid are among these mechanisms. Although these antioxidant defenses eliminate H_2O_2 (and thus superoxide) from the cell, this species can be converted to another highly reactive ROS (•OH) in the presence of reduced transition metals (Cu, Fe) through the Fenton reaction.[18]

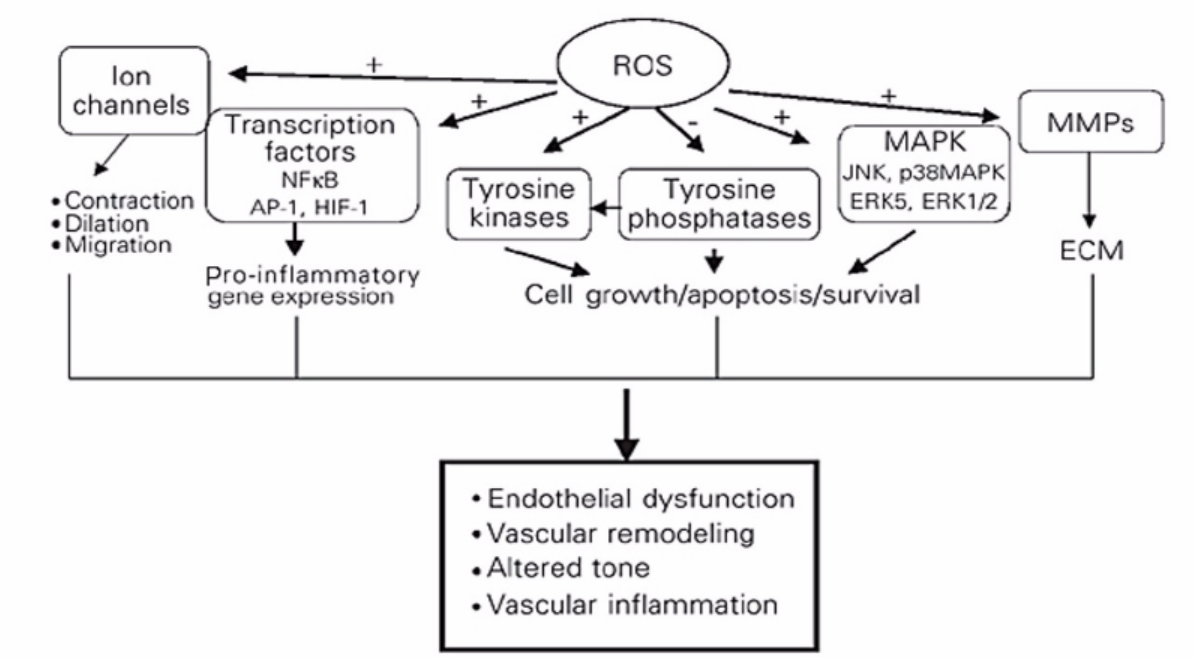


Figure 2: Redox-dependent signaling pathways mediated by Ang II in vascular smooth muscle cells. Intracellular reactive oxygen species (ROS) modify protein tyrosine kinase activity, such as Src, Ras, JAK2, Pyk2, PI3K, and EGFR, as well as mitogen-activated protein kinases (MAPK), particularly p38MAPK, JNK and ERK5. ROS may inhibit protein tyrosine phosphatase activity, further contributing to protein tyrosine kinase activation. ROS also impact on gene expression by activating transcription factors, such as NFκB, activator protein-1 (AP-1) and hypoxia-inducible factor-1 (HIF-1). ROS stimulate ion channels, such as plasma membrane Ca2+ and K+ channels, leading to changes in cation concentration. Activation of these redox-sensitive pathways results in numerous cellular responses that, if uncontrolled, could contribute to hypertensive vascular damage. -, inhibitory effect; +, stimulatory effect; ECM, extracellular matrix; MMPs, matrix metalloproteinases.[32]

pathology for the visualization of oxidative stress phenomena.

- Total antioxidant state quantification. There are several ways to estimate this: spectrophotometry, HPLC, and chemiluminescence, among others.[8,9]

Antioxidants

They are defined as any substance that delays, prevents or removes the oxidative damage from a target molecule.[10] either by stabilizing or deactivating free radicals before they interact with the cells. Antioxidants are a critical component in the optimal maintenance of cell and systemic homeostasis, as well as preserving their health.

Depending on their nature, they can be classified into two main categories:

- Enzymatic, which includes superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase.
- Non-Enzymatic, which includes vitamin E, vitamin C, glutathione, carotenes and retinols, as well as some transition metals (Zn, Cu and Se).[11,12]

Antioxidant systems are highly sophisticated and complex, working interactively and synergistically to neutralize reactive oxygen species. The Antioxidant System has several components, as shown in Table 1.

Previous studies suggest an inverse correlation between food intake or antioxidant plasmatic levels and DNA oxidative damage. Furthermore, vitamin C or E supplementation diminishes lipid oxidative damage, even in smokers with high body mass index, and patients diagnosed with alcohol-induced chronic liver disease, hypercholesterolemia and diabetes. Current evidence suggests that antioxidant supplementation can contribute to decrease oxidative biomarkers in subjects submitted to high stress levels but not in unchallenged subjects.[13]

Antioxidant components in food

There are many food components with antioxidant properties, such as α-tocopherol, γ-tocopherol, tocotrienol, ascorbic acid, β-carotene, and other substances like ubiquinol, and phenolic compounds.[14,15]

Furthermore, nutrition plays a very important role in the maintenance of antioxidant enzymes because some

micronutrients such as selenium, copper, iron, manganese and zinc function as cofactors or are included as part of their prosthetic groups. If micronutrient is insufficient, the enzyme defense system can be impaired.[16]

Antioxidants obtained through diet can act in different ways: first, preventing the excess of free radicals, thus avoiding oxidative damage to the cell. Secondly, after damage has occurred, antioxidants can control free radical levels preventing further damage thereby alleviating some symptoms caused by oxidative stress.[17,18] (Figure 1)

Polyphenols are a heterogeneous group of molecules consisting of aromatic rings substituted with a hydroxyl group. Phenolic compounds are classified in several types depending on their structure. The most important are: phenolic acids and flavonoids (mainly found in vegetables), are found in the surface layers of vegetables, fruits, cereals and other seeds. Their function is to protect the lower layers from oxidation.[19]

These compounds are secondary metabolites produced by plants and they are part of a defense mechanism against ultraviolet radiation and aggression by pathogens. The long-term consumption of plants rich in polyphenols provides protection against cancer, cardiovascular disease, diabetes, osteoporosis and neurodegenerative diseases according to epidemiology studies and meta-analysis. In food, polyphenols contribute to acidity, astringency, color, flavor, odor and oxidative stability. There are also substances with estrogenic activity (phytoestrogens) like isoflavones, lignans and the stilbene resveratrol, whereas others, like tanins, are able to bind to metals and proteins, affecting their bioavailability and leading to some unspecific effects such as antimicrobials or the prevention of neurodegenerative diseases.[20,21]

Bioavailability and metabolism of polyphenols

Bioavailability is the proportion of nutrients that are digested, absorbed, and metabolized through the central metabolic pathways. In addition to polyphenol levels in food, it is important to know the amount of polyphenols that are bioavailable from the total content.[22]

Most polyphenols in foods are present in non-absorbable forms like esters, glucosides or polymers. Before they can be absorbed, they must be hydrolyzed by intestinal enzymes like β-glucosidase and lactase phlorizin hydrolase. Alternatively, they are degraded by the colonic microflora that hydrolyzes glucosides into aglycones that are further metabolized to aromatic acids (such as benzoic acid, equol, enterolactone and enterodiol).[22]

A subset of polyphenols such as quercetin, daidzein or genistein, but not their glycosides, can be absorbed directly through the stomach, just like some anthocyanidins or phenolic acids like chlorogenic acid. However, the rest of the polyphenols arrive intact to the small intestine. Glucosides can be hydrolyzed through two possible routes: the first one involves the aforementioned lactase phlorizin hydrolase (LPH) in the brush border of epithelial cells in small intestine. The activity of this enzyme results in a free aglycone that can passively diffuse through the membranes because it's increased hydrophobicity. The second mechanism involves the cytosolic β-glucosidase in epithelial cells where the polar glucosides are transported by the sodium-glucose transport protein (SGLT1). Although, it has been reported that SGLT1 can transport neither flavonoids nor their glycosylated forms including aglycones, being the latter inhibitors of this transporter. Once absorbed, they undergo methylation, sulfation, or glucuronidation by hepatic enzymes: Catechol-O-methyltransferase (COMT), phenol sulfotransferase (SULT) and UDP-glucuronosyltransferase (UDPGT).[21-23]

In general, those polyphenols that reach the blood and tissues are different from the ones originally present in food. Several in vivo studies suggest that only 5% of the daily total ingested polyphenols are absorbed at the duodenum. From this percentage, only 5%(mainly flavonols) reach bloodstream without any structural changes. The rest of the ingested polyphenols (95%) is directed to the colon, where they are fermented by colonic microflora generating microbial metabolites that are subsequently absorbed appearing as conjugates in plasma. Once absorbed and metabolized, polyphenols can return to duodenum and be transported through enterohepatic circulation, extending their presence within the organism. Finally, before their elimination through urine or bile, plasma-circulating polyphenols bind to albumin. Polyphenols' affinity for this protein depends on its chemical structure. It is unknown whether its function is independent of this mechanism or if this contributes to their activity. Afterwards, they are able to incorporate themselves into tissue, particularly those that underwent metabolic transformation (hepatic tissue, stomach, intestinal, colonic and renal). However, they can also be accumulated in specific target tissues, e.g., pulmonary, pancreatic, brain, cardiac, and splenic.[24,25]

Antioxidant foods and their role in health

Because of the great chemical diversity of antioxidant compounds in food, there is no complete database of their contents. Additionally, antioxidant levels do not necessarily reflect the total antioxidant capacity (TAC), but

Table 2: Methods to assess antioxidant capacity

Type	Assay	Function	Advantages and disadvantages	Reference
	ORAC (oxygen radical absorbance capacity)	It measures the inhibition of oxidations mediated by peroxyl radical and thus it reflects the classical radical chain-breaking antioxidant activity by H atom transfer.	Provides a controllable source of peroxyl radicals that simulate theinteractions between antioxidants and lipids in both food and physiological systems. At critical temperatures (37ºC), small differences can decrease the reproducibility of the assay.	35
	TRAP (total radical-trapping antioxidant parameter)	This method monitors the ability of antioxidant compounds to interfere with the reaction between peroxyl radicals (generated by AAPH or ABAP) and a target probe.	This assay is used for AOC assessment *in vivo*, in serum or plasma because it detects non-enzymatic antioxidants. Its greatest strength is also its drawback: too many different endpoints, comparisons between laboratories are difficult. Additionally, it is complex and time-consuming.	36
HydrogenAtom Transfer (HAT)	*TOSC (total oxidant scavenging capacity)*	This method quantifies the absorbance capacity of antioxidants, specifically when exposed to three strong oxidizing agents: hydroxyl radicals, peroxyl radicals and peroxynitrite. The oxidizable substrate is KMBA, which forms ethylene	It allows antioxidant capacity quantification toward three oxidants. However, the method is not readily adaptable for high-throughput analyses. There is no linear relationship between TOSCinhibition percentagesby the antioxidant and the antioxidant concentration. Comparison between food becomes difficult because multiple endpoints.	37
	CL (Chemiluminescence)	A high-sensitivity modification of the TRAP method. It monitors radical-mediated reactions by CL. CL monitoring is based on the reaction between radical oxidants and specific probes producing excited-states that emit luminescence.	CL reactions are adaptable for automation and can be performed in micro well plate format. The selection of the emitter is a critical consideration. Luminol has been extensively used to study radical-mediated reactions and is acceptable when only one oxidant is being measured. However, the use of luminol in systems containing several oxidants is not advised.	38

	PCL (Photo-chemiluminescence)	It involves the photochemical generation of superoxide free radicals in combination with CL detection. Ascorbic acid and Trolox are typically used as calibration reagents.	This method is not restricted to a specific pH value or temperature range. It is a time-saving and low-cost system for the determination of the integral antioxidative capacity toward superoxide. It is not adaptable to a high-throughput assay system	39
	Croton or β-carotene bleaching by LOO·	Carotenoids undergo bleaching through light- or heat-induced autoxidation.Its oxidation mediated by peroxyl radicals has the same effect. This absorbance loss can be restricted or prevented by classical antioxidants that donate hydrogen atoms in order to quench radicals	This method is adaptable to high-throughput methodology such as microplates. However, temperature control is critical. There are no standard formats to express theobtained results as every study has a different method to calculate inhibition kinetics.	40
	Low-Density Lipoprotein (LDL) oxidation	The oxidation of LDL, freshly isolated from blood samples, is triggered by Cu(II) or AAPH. Lipid peroxidation is monitored at 234nm to detect conjugated dienes. Alternatively, peroxide values are used for lipid hydroperoxides detection.	The use of AAPH as radical is relevant regarding the oxidation reactions that might occur *in vivo*. LDL must be isolated on a regular basis. As this requires blood samplesfrom different individuals, it is not possible to maintain consistent preparations.	41
Single Electron Transfer (SET)	*FRAP (ferric reducing antoxidant power)*	This reaction monitors the reduction of TPTZ forming a colored product. Originally, it was developed to measure reducing power in plasma, but has been adapted to assay antioxidants in botanicals.	FRAP results can be highly variable depending on the time scale.This testconsiders that redox reactions proceed so rapidly that all reactions are completed after 4 minutes, although this is not always the case. Fast-reacting phenols are best analyzed within short time frames. However, some polyphenols react slowly and require longer time frames to allow detection (30 minutes). This test does not measure thiol antioxidants (glutathione). It is simple, fast, inexpensive and robust.	42
	CUPRAC (Copper Reduction assay)	These tests are modifications of the FRAP assay that use Cu instead of Fe. They are based on the reduction of Cu(II) to Cu(I) by the combined action of all antioxidants in the sample	Copper, in contrast to iron,is useful to detect with little interference all types of antioxidants, including thiols. Copper reduction assays are not convenient when the sample consists of a complex antioxidant mixture, as it is difficult to select appropriate reaction time frames.	43

	TEAC (Trolox equivalent antioxidant capacity)	It is based on the antioxidant's ability to scavenge the long-life radical anion ABTS$^{\bullet}$. Results are expressed relative to those obtained using Trolox	The ABTS radical used in TEAC assays is not found in mammalian biology and thus it represents a "nonphysiological" radical source. Additionally, the TEAC reaction may not be the same for the slow reaction, and it may take a long time to reach an endpoint.	[44]
HAT and SET	*DPPH Assay*	The DPPH•radical is one of the few stable organic nitrogen radicals characterized by a deep purple color. This assay is based on the measurement of antioxidants reducing ability toward DPPH.	The test is simple and rapid and requires only a UV-vis spectrophotometer. However, interpretation is complicated when the test compounds have absorption spectra overlapping that of DPPH at 515 nm. This is the case for carotenoids.	[45]
	F-C method (Folin-Ciocalteu) Total Pheolics Assay	This assay has been used as a measure of total phenolics in natural products, but the basic mechanism is an oxidation/reduction reaction.	The method is simple, sensitive, and precise. However, the reaction is slow at acid pH, and it lacks specificity. It suffers from a number of interfering substances (sugars, aromatic amines, sulfur dioxide, ascorbic acid and other enediols and reductones, organic acids and Fe(II))	[46]

AAPH or ABAP: 2,2´-azobis(2-amidinopropane) dihydrocloride, KMBA: α-keto-γ-methiolbutyric acid, TPTZ; ferric 2, 4, 6-tripyridyl-s-triazine, DPPH:2,2-Diphenyl-1-picrylhydrazyl

it also depends on the synergistic and redox interactions between the different food molecules, including their boiavailability. Furthermore, geographic differences should also be considered when regional databases are applicable.[26]

Mexican foods that typically have the highest antioxidant capacity are: zucchini, beetroot, purslane, avocado, peppermint, Chaya (or tree spinach), guava, apple, papaya, orange, prickly pear, watercress, lima beans, punch, mole, boiled corn (esquites), mushrooms, spinach, red enchiladas, spices like cacao, garlic, cinnamon, chili, herb tea (*Dysphania ambrosioides*), cloves, cumin, oregano, annatto, bay leaves, and pepper, among others.[27,28]

Fruits and vegetables contain a wide variety of antioxidant compounds (phytochemicals), such as phenols and carotenoids that protect cells from oxidative damage and decrease the risk of chronic disease. Previous studies established that their consumption contributed to cancer prevention (lung, colon, breast, uterine cervical, esophagus,

Table 3: Total antioxidant capacity (TAC) in food, TEAC method

Group	Food	TAC
Fruits	Guava	28.5
	Apple	27.1
	Papaya	25.1
	Blackberry	20.24
	Orange	20.1
	Plum	14.82
	Apricot	12.8
	Strawberry	10.94
	Banana	8.2
	Lemon	6.7
	Watermelon	2.6
	Fig	5.2
	Raisins	6.6

Vegetables	Zucchini	30.07
	Raw Beetroot	24.92
	Avocado	22.4
	Watercress	21.4
	Chili	19.15
	Lettuce	18.64
	Onion	17.11
	Cooked Beetroot	16.35
	Radish	15.04
	Celery	10.54
	Cucumber	9.54
	Cooked spinach	9.11
	Tomato	8.11
	Carrot	7.69
	Cooked Broccoli	6.42
	Cooked chard	6.63
	Cooked squash	6.24
	Cabbage	2.04
Cereals	Tortilla (corn)	7.11
	Whole-wheat Pasta	5.15
	Oats	4.05
	Cornflour	3.01
	Oatflour	2.79
	Wheatflour	2.7
	White rice	2.2
	Cornflakes	2.19
	Pasta	1.99
Legumes	Length	13.26
	Lentils	9.3
	Green peas	3.73
	KidneyBeans	3.30
	Chickpea	2.9
Oilnuts	Nuts	137.01
	Pistachios	61.46
	Almonds	13.36
	Hazelnuts	12.02
	Pine nuts	5.25
	Peanuts	4.76
Spice, Sweets and beverages	Dark chocolate	94.81
	BayLeaf	47.93
	Rosemary	43.95
	Espressocoffee	36.54
	Milk chocolate	36.16
	Instantcoffee	32.48
	Oregano	30.65
	Groundcoffee	30.29
	Peppermint	27.22
	Sage	23.43
	Basil	21.78
	Hibiscus	12.81
	Red wine	12.14
	Coriander	7.91
	Beer	1.04

Oils	Soy	2.20
	Extra virgin olive oil	1.79
	Corn	1.29
	Sunflower	1.17
	Olive	0.63
	Peanut	0.61

The value indicates the antioxidant capacity in mmol of Trolox equivalents/g. Taken from: Total Antioxidant Capacity of Plan Foods, Beverages and Oils Consumed in Italy Assessed by Three different *in vitro* assays, Total antioxidant capacity of spices, dried fruits, nuts, pulses, cereals and sweets consumed in Italy assessed by three different *in vitro* assays and Capacidad antioxidante total en alimentos convencionales y regionales de Chiapas, México[26,57,58]

oral cavity, stomach, bladder, pancreas and ovary).Low consumers (<500 mg/day) had twice the risk of developing cancer compared to the high consumers (500-1000 mg/day).[29]

Whereas ROS production in normal cells is low and they function as proliferation stimuli, cancer cells display a higher ROS production. Furthermore, ROS are closely linked to tumor development, progression and maintenance. Their higher levels are mediated by a constant activation of transcriptional factors such as NF-κ B and AP-1. Oxidative stress can also induce DNA damage leading to genomic instability resulting in cancer progression. Some factors that relate cancer and ROS are: growth factors such as EGF and PDGF (cells stimulated by them increase ROS production), Ras mitogenic activity depends on superoxide, Nox1 overexpression (NADPH oxidase catalytic subunit) induces both superoxide generation and cell transformation enhancing tumor aggressiveness. High ROS levels have also been linked to MAPK (mitogen-activated protein kinases) activation enabling malignant progression in several cell lines.[30]

Activation of the Angiotensin II receptor stimulates non-phagocytic NADPH oxidase concomitantly with the generation of both superoxide and H_2O_2 in several vascular cell types, including vascular smooth muscle cells, endothelial cells and fibroblasts. Intracellular ROS modify the activity of tyrosine kinase proteins as well as that of MAPK. They also inhibit tyrosine phosphatase proteins, strengthening the signals mediated by tyrosine kinases. Figure 2 shows ROS influence on several factors that regulate vascular performance. Regarding the prevention of cardiovascular disease, flavonoid consumption has been inversely correlated to plasmatic cholesterol and LDL (low density lipoprotein), counteracting the appearance of arterial coronary disease. Conversely, the role of carotenoids as anti-cancer supplements has recently been questioned according to the results observed in several clinical studies. In one of these, it was observed that the incidence of non-melanoma skin cancer was indistinguishable from that of patients receiving a β-carotene supplement. Other study showed that smokers gained no benefit from supplemental β-carotene regarding lung cancer incidence and even a deleterious effect was suggested.[31,32]

Antioxidants are chemical substances characterized for preventing or delaying the oxidation of several substances, mainly fatty acids. These reactions take place in both food and human organisms and they are the underlying cause of major physiological alterations that lead to pathological states. Another function of antioxidants is to allow the use of physiological oxygen by mitochondria, avoiding the effects of oxidative stress and the lack of oxygen. This is achieved through the formation of complexes that inhibit the reactions that produce oxidant radicals. Therefore, antioxidants play an important role in non-transmissible chronic diseases.[33]

In order to measure antioxidant capacity some experimental techniques were developed, some of these are listed below:

Methods to assess antioxidant capacity in food

Antioxidants deactivate a radical by two independent mechanisms: Hydrogen Atom Transfer (HAT) or Single Electron Transfer (SET). They both result in radical elimination.

The methods used to evaluate antioxidant capacity have the following requirements: a) they monitor chemical reactionsthat actually occur in potential application(s), b) they use a biologically relevant source of radicals, c) they are simple, d) they use a method with a defined endpoint and a previously identified chemical mechanism, e) the instrumentation is readily available, f) they must possess good reproducibility within-runs and among day-to-day experiments, g) they must be adaptable for the assay of both hydrophilic and lipophilic antioxidants and also they must use different radical sources, h) they must be adaptable to "high-throughput" configurations for routine quality control analyses.[34] Some methods along with their descriptions are presented in Table 2.

The First International Congress on Antioxidant Methods of 2004 consider the standardization of three assays: 1) the ORAC assay that represents the HAT reaction mechanism and is considered the most important in human biology, 2) the Folin-Ciocalteau method, a SET based assay that quantifies reduction capacity, which is normally used to

expresses phenolic content, widely used in the botanical field, and 3) TEAC, another method based on SET commonly used to measure antioxidant capacity in food, beverages and nutritional supplements.[34]

A diet rich in fruits, vegetables and minimally refined cereals is associated with lower risk for chronic degenerative diseases, it has been assumed that dietary antioxidants may explain this protective effect. Table 3 shows various foods and their total antioxidant capacity based on the TEAC method (mmol equivalent Trolox/g).[56]

There are no guidelines in North or Latin America that indicate the Daily Recommended Intake of antioxidants and polyphenols. The US Department of Agriculture and the Department of Health and Human Services, in the Dietary Guidelines (2010), advises theconsumption of a similar amount to the daily recommendation of Vitamin C (1000 mg/day). European populations ingest 5 or more portions of fruits and vegetables daily, reaching this recommended intake value. This is associated with a lower risk of non-transmissible diseases.[29]

CONCLUSION

Food contains several components with important antioxidant capacity. Endogenous antioxidants constitute a complex system and protect them from highly reactive and oxidative molecules such as reactive oxygen species, preventing the development of several diseases and the prevention of premature aging.

ACKNOWLEDGEMENT

Partially supported by CIC-UMSNH 16.2-2015.

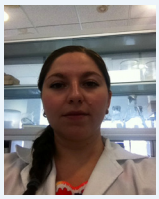
CONFLICT OF INTEREST

The authors declare no conflict of interest.

Highlights of the Paper

- In this review, some basic concepts of reactive oxygen species, oxidative stress and antioxidants are analysed.
- Antioxidant foods and their role in health
- Some of the methods used to assess the antioxidant capacity of foods are included.
- Some of the food highly consumed by the Mexican population has antioxidant capacity and it is herein summarized.

About Authors



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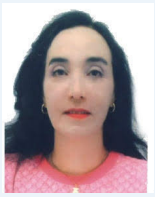
México. She has about 15 participations in congress from the past 3 years.



PhD. Bertha Fenton Navarro: *Presently working as Research-Professor in the Department of Postgraduate studies at the Medicinal Faculty of the University UMSNH (Universidad Michoacana de San Nicolás de Hidalgo).*
She has 25 publications in the JCR (Journal Citation Reports), 50 within index journals. 2 book chapters. 27 graduate students (bachelor and postgraduate), more than 50 postgraduate courses. Several invited talks. More than 100 participation in congress. She belongs to different scientific societies.

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