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Review Article

ANTIOXIDANTS TO THE RESCUE OF CELL UNDER INVASION OF FREE RADICALS – A REVIEW

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ABSTRACT
Free radicals as superoxides, hydrogen peroxide, nitric oxide, hydroxyl groups have incriminated as culprits for initiating or promoting cellular damage of various degrees. The present review focuses on an insight of free radicals and antioxidants, their sources, types, possible mechanisms of action in human health and disease. The review also explores the positive role of free radicals in immunity and phagocytosis. Different types of antioxidants which inactivate free radicals have been described. The review also includes the synthetic and naturally occurring antioxidants which are frequently used to negate the harmful effects of free radicals.

Key Words: *Free Radicals, Antioxidants, ROS, Oxidative Stress*

INTRODUCTION
Free radicals and related species have attracted a great deal of attention in recent years. Wherever there is disease or destruction, there are free radicals. Wherever there is life, there are free radicals. Free radicals are defined as atoms or molecules with one or more unpaired electrons (Vallyathan and Shi, 1997) which makes them highly reactive (Takimoto and Kass, 2007) . They are molecules or molecular fragments containing an unpaired electron in the valence shell and are capable of independent or free existence (Sen, 1995).The most common free radicals in the body are created from oxygen which is akin to body's energy vending machine for funding every metabolic transaction. It is true the human body must metabolize a great deal of oxygen while exercising. However a little overdone the same mechanism causes production of these free radicals.

Free radicals always tend to chemically react with other molecules, such that an electron could be stolen from the attacked molecule in order to pair the radical's unpaired electron. Due to this tendency they usually react with cellular macromolecules including DNA, proteins and lipids, and interfere with vital cellular functions (Vaughan, 1997). The same oxidative process also causes oils to become rancid, peeled apples to turn brown and iron to rust.

Free radicals containing oxygen are known as reactive oxygen species (ROS) and are the most biologically significant free radicals. They represent a broad category of radical (hydroxyl ion, superoxide, nitric oxide, peroxyl etc) and non radical (ozone, singlet oxygen, lipid peroxide, hydrogen peroxide) oxygen derivatives (Aggarwal and Prabakran, 2005) Reactive nitrogen species nitrous oxide, peroxyl nitrite, nitroxyl ion etc. are also a class of free radicals derived from nitrogen and considered as a subclass of ROS (Sikka, 2001).

Exposure of the cell membranes to oxygen radicals stimulates the process of lipid peroxidation in which poly unsaturated fatty acids are degraded to a variety of aldehydes(i.e. malonaldehyde, propranolol, hexanal etc) (Dargel, 1992). Leading to a disturbance of membrane integrity (Hemnani and Parihar,1998).

Free radicals, however, are not always harmful. In the living system they are necessary for cell maturation. Also free radicals released by WBCs destroy invading pathogenic microbes and help in body's defense against diseases. Hence the complete elimination of free radicals would not only be impossible, but also harmful (Bagchi and Puri, 1998). In other words free radicals are integral part of metabolism and are formed continuosly in the body (Fehrenbach and Northoff, 2001). So it is impossible to avoid damage by free radicals and they are a constant companion of life.

Types of Free Radicals

1. *Superoxide*: It is made by adding one electron to the oxygen molecule. It can be further reduced to hydrogen peroxide, hydroxyl radical (OH) and finally to water (Sen, 1995). Activated phagocytes (neutrophils, monocytes, macrophages, eosinophils) form superoxide which helps in killing foreign organisms (Halliwell, 1994).

2. *Hydroxyl:* The .OH is the most toxic of the oxygen-based radicals and it wreaks havoc within cells, particularly with macromolecules (Reiter *et al.,* 1995). It is short lived but most damaging radical in the body.

3. *Hydrogen Peroxide*: Hydrogen peroxide is not a free radical but falls in the category of reactive oxygen species. It is an oxidising agent and is the main source of hydroxyl radicals in the presence of transition metal ions. It is also involved in the production of HOCl by neutrophils.

4. *Nitric Oxide (NO)*: Nitric oxide is another physiological free radical which is made by vascular endothelium as a relaxing factor, and also by phagocytes and in the brain. It has many important physiological functions but excess can be toxic (Halliwell, 1994).

It is known to be involved in various age related diseases like atherosclerosis, hypertension etc. and in many other biological effects such as blood vessel dilatation , signaling neurotransmission , regulation of hair follicle activity,and immune response . Increased NO may contribute to the development of oxidative stress during aging (Maurya and Rizvi, 2009).

Sources of Free Radicals

Free radicals and other reactive oxygen species are derived either from normal essential metabolic processes in the human body or from external sources such as exposure to x-rays, ozone, cigarette smoking, air pollutants and industrial chemicals (Bagchi and Puri, 1998).

Endogenous Sources

1. *Mitochondria*: Mitochondria are a major source of ROS generation during normal metabolic processes. The respiratory chain is considered as a major source of free radicals. Cellular oxidative phosphorylation results in the univalent reduction of oxygen and the generation of ROS (Sen, 1995).

2. *Microsomes*: Are the second major endogenous source of ROS generation, producing $O_2^{\cdot -}$ and H_2O_2 during normal electron transport reactions. Two additional major sources of production of $O_2^{\cdot -}$ are autooxidation of P_{450} and oxidation of NADPH by NADPH dehydogenase (Vallyathan and Shi, 1997).

3. *Enzymes or Enzymatic Reactions*: Several enzymes generate superoxide in the cell. In hypoxia, the oxidation of xanthine and hypoxanthine by xanthine oxidase results in the generation of $O_2^{\cdot -}{}_{(SUPEROXIDE)}$, which leads to cell injury **.** The tryptophan dehydrogenase in the liver can generate $O_2^{\cdot -}{}_{(SUPEROXIDE)}$ by its specific reaction with tryptophan. Also aldehyde oxidase can react with variety of substrates in the liver to produce $O_{2\,(SUPEROXIDE)}$ other enzymes which can produce $O_2^{\cdot -}{}_{(SUPEROXIDE)}$ are galactose oxidase, mono amine oxidase, cyclooxygenase and lipooxygenase (Vallyathan and Shi, 1997).

4. *Phagocytes*: Phagocytes generate ROS when they come in contact with microorganisms, inhaled particles or other stimuli. Activation of phagocytes triggers respiratory burst, characterized by an increase in oxygen uptake, glucose metabolism and utilization of NADPH. This reaction is catalysed by plasma membrane bound oxidase, leading to increased production of ROS like $O_2^{\cdot -}{}_{(SUPEROXIDE)}$ H_2O_2 and OH (Vallyathan and Shi, 1997).

***5*. *Metal Ions*:** Transition metals especially iron play an important role in generation of ROS. Iron can facilitate the decomposition of lipid peroxides to aldehydes and hydrocarbons. Acidic pH and citrate by causing release of bound iron, promote enhanced ·OH generation (Vallyathan and Shi, 1997). Other transition metals such as As(V), Be(II), Cd(II), Co(II), Cu(II), Hg(II), Pb(II), and Ni(II) also promote free radical reactions.

The oxidative role of transition metals provides growing evidence of metal-induced carcinogenesis. Metals may also contribute to the underlying pathogenic mechanism by promoting inflammation, inhibiting antioxidant defenses, inhibiting DNA repair, and enhancing lipid peroxidation (Vallyathan and Xianglin Shi, 1997).

Exogenous Sources

1. *Cigarette Smoke*: It is the single most avoidable health hazard and is known to cause many diseases. It is a complex mixture of more than 4000 substances (Becker, 1995).A single puff of cigarette smoke contains trillions of free radicals **.**Cigarette smoke literally burns away the antioxidant vitamins C and E, as well as other nutrients (Kidd, 1997). In the lungs cigarette smoke can release H_2O_2 which can react with iron in the nuclei of cells to produce ·OH. ·OH. causes DNA strand break and base damage, which can lead to oncogene activation and thereby promote carcinogenesis (Vallyathan and Shi, 1997).

2. *Alcoholism*: Ingestion of ethanol causes increased lipid peroxidation and also its metabolism produces acelaldehyde that is known to consume GSH (Sen, 1995).

3. *Toxins and Drugs*: Toxins can cause oxidative stress either by increasing free radical activity or by depleting antioxidant defenses.The side effects of several drugs are due to increased oxidative damage.e.g phenylbutazone, nitrofurantoin, pencillamine can be metabolized to generate toxic free radicals and doxorubicin's major side effects (especially cardiotoxicity) may involve excess of superoxide and hydroxyl radical production and also metabolism of paracetamol by liver cytochrome P- 450 generates a product that reacts with and removes glutathione. Loss of glutathione causes secondary oxidative damage, which contributes to hepatic failure in paracetamol overdose (Halliwell, 1994).

4. *Ionizing Radiation*: Particulate type of radiation can disrupt atoms or molecules in the cell, producing free radicals and ions by the direct effect. DNA being most vulnerable, changes induced is DNA strand breaks, base changes, bond cleavage of sugars, degradation and cross linking of DNA. These can also cause mitosis, mutations, and chromosome aberrations in the cell. Indirect effects of radiation to the cell result in the radiolysis of cellular water and formation of free radicals (Vallyathan and Shi, 1997).

Antioxidants

If free radicals are not inactivated, their chemical reactivity can damage cellular macromolecules including proteins, lipids, carbohydrates and nucleic acids (Bagchi and Puri, 1998), fortunately, antioxidants can come to the rescue. Antioxidants means against oxidation, Antioxidants work to protect lipids from peroxidation by free radicals (Dekkers *et al.*, 1996) and can be defined as a molecule stable enough to donate an electron to a free radical and neutralize it, thus reducing its capacity to damage (Bagchi and Puri, 1998). In other words antioxidants are sacrifice molecules that prevent and minimize free radical attack on cell and tissues.

A crucial balance between free radical production and antioxidant defense helps in disease prevention (Semra Sardas, 2003).Under physiological conditions ROS concentrations are kept low by endogenous oxidant enzymes such as superoxide dismutase , catalase, glutathione peroxidase and also by non enzymatic components such as vitamin C, vitamin E, glutathione and uric acid (lyer and Lindsey, 2009). When the production of free radicals is beyond the protective capability of the antioxidant defenses, condition known as oxidative stress occurs. So oxidative stress has been defined as the loss of balance between free radical or ROS production and antioxidant systems with negative effects on carbohydrate, lipid and proteins, thus playing a role in cardiovascular diseases, cancer, diabetes and neurodegenerative disorders etc (Bhogade *et al.*, 2009).

A certain amount of oxidative damage takes place even under normal conditions, however the rate of this damage increases during the ageing process, as the efficiency of antioxidative and repair mechanisms decrease (Maurya and Rizvi, 2009). One of the important markers of oxidative stress (OS) is malonaldehyde (MDA) which is an end product of lipid peroxidation (Das *et al.*, 2009).

Broadly, the possible mechanisms by which antioxidants may protect against ROS are:

1) Prevention of ROS formation.
2) Interception of ROS attack by scavenging the reactive metabolites and converting them to less reactive molecules and/or by enhancing the resistivity of sensitive biological targets to ROS attack.
3) Facilitating the repair of damage caused by ROS.
4) Providing a favourable environment for the effective functioning of other antioxidants (Sen, 1995).

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Classification of Antioxidants

Antioxidants available for therapeutic use are conveniently divided as:

A) Natural antioxidants (Physiological) - Normally present in the body

B) Synthetic antioxidants

Within each group three categories of antioxidants have been recognized.

1) Enzymatic antioxidants that catalyze the breakdown of free radicals

2) Preventive Antioxidants that sequester the metal ions and prevent their participation in free radical generation.

3) Free radical scavengers or chain breaking antioxidants.

1) *Enzymatic Antioxidants*:

a) *Superoxide Dismutases*: SOD' s are indicated to be present in all oxygen metabolizing cells .The principal function of SOD is to catalyse the conversion of superoxides to H_2O_2 which is substantially less toxic than superoxide (Bernhardt *et al*., 2009).

b) *Catalase*: Is mostly localized in the peroxisomes of the liver and kidney, and in microperoxisomes found in other cells (Sen, 1995). It degrades hydrogen peroxide to water and oxygen, and hence finishes the detoxification reaction started by SOD.

c) *Glutathione*: It is the most abundant intracellular thiol and low molecular weight tripeptide found in living cells. It reacts with the free radicals and can protect cells from singlet oxygen, hydroxyl radical and superoxide radical damage (Gupta *et al.,* 2009). Depletition of GSH renders the cells more susceptible to oxidative stress (Das *et al.,* 2009). It also maintains vitamin C and vitamin E in their reduced form, which also exert an antioxidant effect by quenching free radicals (Singh *et al*., 2004).

d) *Hemeoxygenase*: HO plays a critical role in attenuating the production of ROS through its ability to degrade heme in an enzymatic process that leads to the production of equimolar amounts of CO and biliverdin/ bilirubin and the release of free iron (Abraham *et al.,* 2009).

e) *NADPH*: quinone oxidoreductase (NQO_1) catalyzes the two-electron reduction of quinones to hydroquinones thus protecting the cells against oxidative stress (Bruge *et al.,* 2008).

f) *Carbonic Anhydrase*: (carbonic anhydrase III (CAIII) functions as an anti-oxidant agent in skeletal muscle (Zimmerman *et al.,* 2008). and it may be important regulator involved in the anti-lipid and antioxidant effects of lycopene **(**Yuan-Man *et al.,* 2008).

2) *Preventive Antioxidants*

Transition metal ions such as iron and copper containing unpaired electrons readily participate in free radical reactions (Halliwell and Gutteridge, 1989). Metal binding proteins function by inactivating transition metal ions that otherwise would have catalyzed the production of free radicals (Makker *et al.,* 2009). In the presence of transition metal ions, ascorbic acid loses its antioxidant properties and hence absence of such free transition metal ions allows effective antioxidant action of ascorbic acid (Sen, 1995). Therefore, under normal circumstances many endogenous ligands prevent participation of iron and other common transition metals in the generation of ROS in living cells (Vallyathan and Shi, 1997).

Preventive Antioxidants are:

a) *Ferritin*: Is the usual storage form of iron and can not stimulate free radical reactions (Halliwell and Gutteridge, 1989). It has been found in liver, spleen and bone marrow.

b) *Ceruloplasmin*: A copper containing protein is an important intravascular endogenous antioxidant which protects the vascular tunica intima against free radicals (Jaiswal and Saxena, 2009).

c) *Transferrin*: Transferrin in the blood is usually loaded to about 30% capacity so that free iron in the plasma is maintained at a very low level.

d) ***Lactoferrin:*** An iron binding glycoprotein present in milk(Conneely, 2001),scavenges free iron, helping to prevent uncontrolled iron based free radical reactions, thus protecting certain cells from peroxidation.

e) ***Coenzyme Q 10*****:** Also known as ubiquinone is a highly reactive antioxidant which protects biological membranes against oxidation (Sen, 1995).

f) ***Albumin*****:** It represents a very abundant and important circulating antioxidant in plasma .It is well known for its ability to bind metals ions, fatty acids, drugs, and also hormones (Roche *et al.,* 2008). More than 70% of the free radical-trapping activity of serum is due to human serum albumin (HSA) (Bourdon and Blache, 2001). Another aspect of antioxidant activity of albumin may come from its capacity to bind amino acid homocysteine, elevated level of whom is a well-known risk factor for atherosclerosis and may act through oxidation of LDL (Papatheodorou and Weiss, 2007).

g) ***Desferoxamine*****:** It is an important iron chelating agent. It also has anti-oxidant potential as it chelates ferric iron in various parts of the body (Niihara *et al.*, 2002).

h) ***Bcl-2*****:** Is a proto-oncogene shown to intercept the generation of ROS (Sen, 1995).

3. ***Chain Breaking Antioxidants***

a) ***Vitamin E*****:** It is the most powerful lipophilic antioxidant in humans. It contributes to membrane stability (Bhogade *et al.*, 2009) and its supplementation reduces MDA levels , as it protects the membrane lipids and LDL against free radical mediated lipid peroxidation ,thus playing a significant role in reduction of cardiovascular diseases (Jaiswal and Saxena, 2009). Baseline level of MDA ,major end product of lipid oxidation, which is high in smokers, is decreased and activities of the antioxidant enzymes SOD, glutathione peroxidase (Gpx), and catalase (CAT) which are low in smokers, are increased by supplementation of almonds which are rich source of vitamin E (Li *et al.*, 2007).

b) ***Vitamin C*****:** It is a potent water-soluble antioxidant in humans (Padayatty, 2003). It readily donates electrons to break the chain reaction of lipid peroxidation. The water-soluble properties of vitamin C allow for the quenching of free radicals before they reach the cellular membrane.

Uric acid – Has been found to act as an important non enzymatic antioxidant in the blood as well as in other body fluids like seminal plasma. It maintains the cell membrane integrity by inhibiting peroxidation of membrane lipids and also helps to stabilize the antioxidant activity of ascorbic acid in the seminal plasma (Das *et al.*, 2009).

c) ***Melatonin:*** Is the chief secretory product of the pineal gland and synthesized enzymatically from serotonin (5-HT) (Gülçin, 2008). It is a potent endogenous free radical scavenger. It also synergizes with vitamin C, vitamin E and glutathione in the scavenging of free radicals.and has been detected in vegetables, fruits and a variety of herbs**.** Melatonin, once oxidized, cannot be reduced to its former state because it forms several stable end-products upon reacting with free radicals. Therefore, it has been referred to as a terminal (or suicidal) antioxidant (Tan *et al.*, 2000). Low levels of endogenous melatonin production among older individuals may lead to higher levels of oxidatively damaged guanine in DNA, thereby increasing the risk of developing cancer (Davanipour *et al.*, 2009).

d) ***Bilirubin*****:** It is a powerful chain breaking antioxidant in biological systems (Stocker, 2004) and is a prominant endogenous antioxidant cytoprotectant (Sedlak, 2009) Risk of atherosclerosis has been shown to vary inversely with serum levels of bilirubin (Novotný and Vítek, 2003).

e) ***Flavonoids*****:** A number of other dietary antioxidants exist beyond the traditional vitamins collectively known as phytonutrients or phytochemicals .one example is flavonoids, a group of polyphenolic compounds. Good sources of flavonoids include all citrus fruits, berries, onions (Slimestad *et al.*, 2007) pulses (Ewald *et al.,* 1999), tea, red wine and dark chocolate. Flavonoids have been referred to as nature's biological response modifiers because they show anti-allergic, anti-inflammatory (Yamamoto and Gaynor, 2001) anti-microbial (Cushnie and Lamb, 2005) and anti-cancer activity (de Sousa, 2007).The mechanism of antioxidant activity can be characterized by direct scavenging of oxygen free radicals as well as inhibition of oxidative enzymes that generate these ROS (Terao, 2009).

The yellow curry pigment curcumin is a potent phenolic antioxidant (Gupta *et al.,* 2009). Neuroprotective effect of curcumin which is attributed to its antioxidant activity, in animal models of cerebral ischemia has been demonstrated in recent studies (Thiyagarajan and Sharma, 2004).

Drugs in Common Use Having Potent Antioxidant Activity:

***Carotenoids*:** are natural pigments which are synthesized by plants. Most of the carotenoids present in food have antioxidant activity. The abundant ones are alpha-carotene, beta-carotene, lutein, lycopene, cryptoxanthin and zeaxanthin. Beta carotene is most common carotenoid in fruits and vegetables (Sergio *et al.,* 1999). It is a precursor to vitamin A (retinol) . Food sources of these compounds include a variety of fruits and vegetables, although the primary sources of lycopene are tomato and tomato products. Lycopene is known to decrease cardiovascular risks.It decreases total cholesterol, LDL, the plasma malonaldehyde levels and increases serum HDL levels. The increased activities of SOD, catalase and glutathione peroxidase further support the antioxidant effects of the lycopene (Yuan-Man *et al.,* 2008). Additionally, egg yolk is a highly bioavailable source of lutein and zeaxanthin (Johnson, 2002).

***Estrogens*:** Free-radical scavenging by estrogens has been implicated as one of their non-genomic mechanisms of cytoprotection. Direct oxyradical-scavenging by estrogens has been thought to cause interruption of free-radical chain reactions such as lipid peroxidation, and to prevent free-radical induced oxidative damage to macromolecules such as DNA and proteins (Prokai *et al.,* 2006). Furthermore, estrogens can induce antioxidant enzyme expression by stimulating the antioxidant defense system (Barp *et al.,* 2002).

***Probucol*:** Is an anti-hyperlipidemic drug (Yamamoto, 2008). It is a potent anti-oxidant drug that has been in clinical use for the treatment and prevention of cardiovascular diseases. Probucol and antioxidant vitamins rescue cigarette smoke-dependent impairment of ischemia-induced neovascularization. The mechanisms involve beneficial effects on oxidative stress levels in ischemic tissues together with an improvement of endothelial progenitor cells functional activities (Turgeon *et al.,* 2010).

***Pycnogel*:** Pycnogenol is a trademarked name for proanthocyanidin, a bioflavanoid complex extracted from the bark of the European coastal pine, Pinus Maritima. It has been shown to have antioxidant and free radical scavenging activitie (Gandin *et al.,* 2009). It improves the legs'heaviness, subcutaneous edema and reduces venous pressure in chronic venous insufficiency probably by either stabilizing the collagenous subendothelial basal membrane or scavenging the free radicals, or by a combination of these activities. Pycnogenol can be therefore recommended both for prevention and treatment of CVI and related veno-capillary disturbances (Cesarone *et al.,* 2006 a). It improves diabetic microangiopathy thus helps in preventing diabetic ulcerations (Cesarone *et al.,* 2006b). Also pycnogenol treated patients of retinopathy show an improvement of retinal vascularization and a reduced endothelial permeability and leakage resulting in significant recovery of visual acuity (Spadea and Balestrazzi, 2001).

***Lazaroids*:** The 21-aminosteroid compounds are potent lipid per-oxidation inhibitors belonging to a new class of anti-oxidants given the collective name of lazaroids (Campo *et al.,* 1997). They possess the membrane-stabilizing effects of the glucocorticoids without the receptor- dependent side effects. One of these compounds, tirilazad mesylate (U-74006F) has been selected for clinical development as a parenteral neuroprotective agent. They exert their anti-lipid peroxidation action through two mechanisms, free radical scavenging and membrane stabilization. Their antioxidant activity is 100 times more potent than corticosteroids and therefore may be efficacious in the clinical management of acute CNS injury (Kavanagh and Kam, 2001).

***Urosolic Acid*:** It inhibits various types of cancer cells by inhibiting the STAT3 activation pathway (Pathak *et al.,* 2007) and human fibrosarcoma cells by reducing the expression of matrix metalloproteinase-9 (Shishodia *et al.,* 2003) by acting through the glucocorticoid receptor. It is present in many plants, including apples, bilberries and peppermint.

***Spirulina*:** Is microscopic blue - green algae containing an unusually high amount of protein. Administration of spirulina has been found to increase the total antioxidant level (Park *et al.,* 2008).

***Adenosine*:** Adenosine decreases superoxide generation by neutrophils and also affects endothelial function. It also protects against reoxygenation injury (Sen, 1995).

Silymarin: It is a polyphenolic flavonoid antioxidant extracted from Silybum marianum (milk thistle), an edible plant. Hepatoprotective effect in both acute and chronic hepatitis is accomplished by antioxidative and lipid peroxidation inhibition mechanisms (Dixit *et al.,* 2007). It is found to be useful also in treatment and prevention of many neurodegenerative and neurotoxic processes, gastrointestinal problems and cardiotoxicity (Wang *et al.,* 2002; Soto *et al.,* 2003; Chlopovkova *et al.,* 2004).

***Alpha Lipoic Acid*:** Is a disulfide compound produced in small quantities in cells (Evans and Goldfine, 2000). It can be found in very small amounts in foods such as spinach, broccoli, peas, brussel sprouts, rice bran. It has been found to be useful in the treatment of alzheimers disease as it increases acetylcholine (ACh) production by activation of choline acetyltransferase and increases glucose uptake, thus supplying more acetyl-CoA for the production of Ach (Holmquist *et al.,* 2006). Diabetic polyneuropathy is improved by oral treatment with alpha lipoic acid (Ziegler *et al.,* 2006). As an antioxidant, LA directly terminates free radicals, chelates transition metal ions, increases cytosolic glutathione and vitamin C levels and prevents toxicities associated with their loss (Smith *et al.,* 2004).

***Trace Elements*:** They are involved in gene expression, RNA and DNA metabolism, cellular immune functions, and also play a role in cellular protection against free radical injury (Khanna *et al.,* 2009). Selenium has anticarcinogenic effect as it prevents the malignant transformation of normal cells and the activation of oncogenes. So Selenium supplementation should start early in life and be maintained over the entire lifespan (Schrauzer, 2000). Zinc supplementation is associated with decreased oxidative stress and improved immune function, which may be among the possible mechanisms for its cancer preventive activity (Prasad and Kucuk, 2002).

CONCLUSION

The knowledge of the mechanisms of how free radicals lead to cellular damage is imperative before employing methods or mechanisms to inhibit their activity. Several iatrogenic options are now available which use natural sources of antioxidants or generate them using modern technology and principles of biotechnology. The review introduces the commonly used drugs and sources of antioxidants to minimize cellular damage.

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