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RESEARCH ARTICLE

LYCOPENE- AN ANTI-OXIVORE WITH WEALTH OF BENEFITS

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ABSTRACT

Over the past few years, immense importance has been given to free radicals and anti-oxidants in the field of dentistry. Depending on the balance between their destruction and formation, free radicals can either be beneficial or deleterious to health. Stabilization of free radicals is done by anti-oxidants, thus preserving the cells from destruction. Hence, antioxidants that are prolific in our diet holds the interest of today's dental community as an armoury to positively impact oral environment. One such anti-oxidant is lycopene. Lycopene, the red pigment of ripe tomatoes, watermelons, red chillies and guavas, is a tetraterpene with eight isoprene units composed entirely of carbon and hydrogen. This highly unsaturated hydrocarbon contains 11 conjugated and two nonconjugated carbon–carbon double bonds. It is a strong antioxidant and free radical-scavenger because it possesses the property of quenching singlet oxygen. This review article is an overview of various anti-oxidants in general and lycopene in particular.

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INTRODUCTION

Oxygen is an element indispensable for life. It can promote and deteriorate heath of the body. In 1954 Gershman gave radical theory of oxygen toxicity and since then the poisonous effects of oxygen were noticed. Thus theory states that the toxicity of oxygen is due to partially reduced forms of oxygen. When cells use oxygen to generate energy in the form of ATP in the mitochondria, free radicalsare created. Free radicals are chemically active atoms that have a charge due to an excess or deficient number of electrons. They can be reactive oxygen species (ROS) or reactive nitrogen species (RNS) (Shyam Patel, 2015). Reactive oxygen species are highly reactive oxidant molecules that are generated endogenously through regular metabolic activity, lifestyle activity and diet. They react with cellular components, causing oxidative damage to such critical cellular biomolecules as lipids, proteins and DNA (Sanjiv Agarwal, 2000). ROS and RNS are well recognized for playing a dual role as both deleterious and beneficial species.[1] ROS are highly reactive molecules derived from oxygen metabolism. In vivo, some of these ROS play constructive roles in cell physiology; however, they may also cause great destruction to cell membranes and DNA, by causing membrane lipid peroxidation, decreased membrane fluidity, and DNA mutations leading to cancer, degenerative, and other diseases (Harman, 1994). At low or moderate levels, ROS participate in the biosynthesis of molecules such as thyroxin, prostaglandin and are used by the immune system. Macrophages and neutrophils generate ROS in order to kill the bacteria that may engulf by phagocytosis (Schreck, 1991). At high concentrations; they generate oxidative stress and nitrosative stress, a deleterious process that can damage all cell structures (Genestra, 2007). ROS levels increase intensely and result in significant damage to cell structures during oxidative stress. ROS are cytotoxic and have been implicated in the aetiology of various human diseases (Chapple, 1997). Thus, ROS can kill bacteria but it also destroys the adjacent infected host tissues (Shyam Patel, 2015). The human body has several mechanisms to counteract oxidative stress by producing antioxidants which are either naturally produced in body, or externally supplied through foods and /or supplements. In a normal cell there is balance between formation and removal of free radicals. However, this balance can be shifted towards more formation of free radicals or when levels of antioxidants are diminished. This state is called 'oxidative stress' & can result is serious cell damage if the stress is massive and prolonged (Vasundhara Shivanna, 2013).

History: In 1900, Gomberg recognized organic free radicals, which lead to the speculation that free radical species might be involved in living system. In 1954, Gerschman and colleagues proposed for the first time that damaging effects of oxygen could also be attributed to the formation of oxygen free radicals. In 1956, Harman proposed the free radical theory of

ageing. The interest in this theory of ageing was reawakened only 13 year later when in 1969, McCord and Fridovich identified superoxide dismutase, the first enzymatic defense mechanism against the superoxide anion an important free radical, thus a new arena of research began. Britton chance group in Philadelphia first indicated the role of cellular mitrochondrial respiration to generate reactive oxygen species which was supported by Nohl and Hegner in (1978). Halliwell and Gutteridge in 1989 reported reactive oxygen species include both free radicals as well as non-radical derivatives of oxygen. They also defined antioxidants for the first time. Sen, in 1995 defined oxidative stress and at present number of researchers have documented the role of free radicals and reactive oxygen species in number of pathophysiological states (Vishal Tandon, 2005).

Sources of free radical insults in dental therapy

In dentistry, many commonly used dental materials may form free radicals like (Vasundhara Shivanna, 2013)

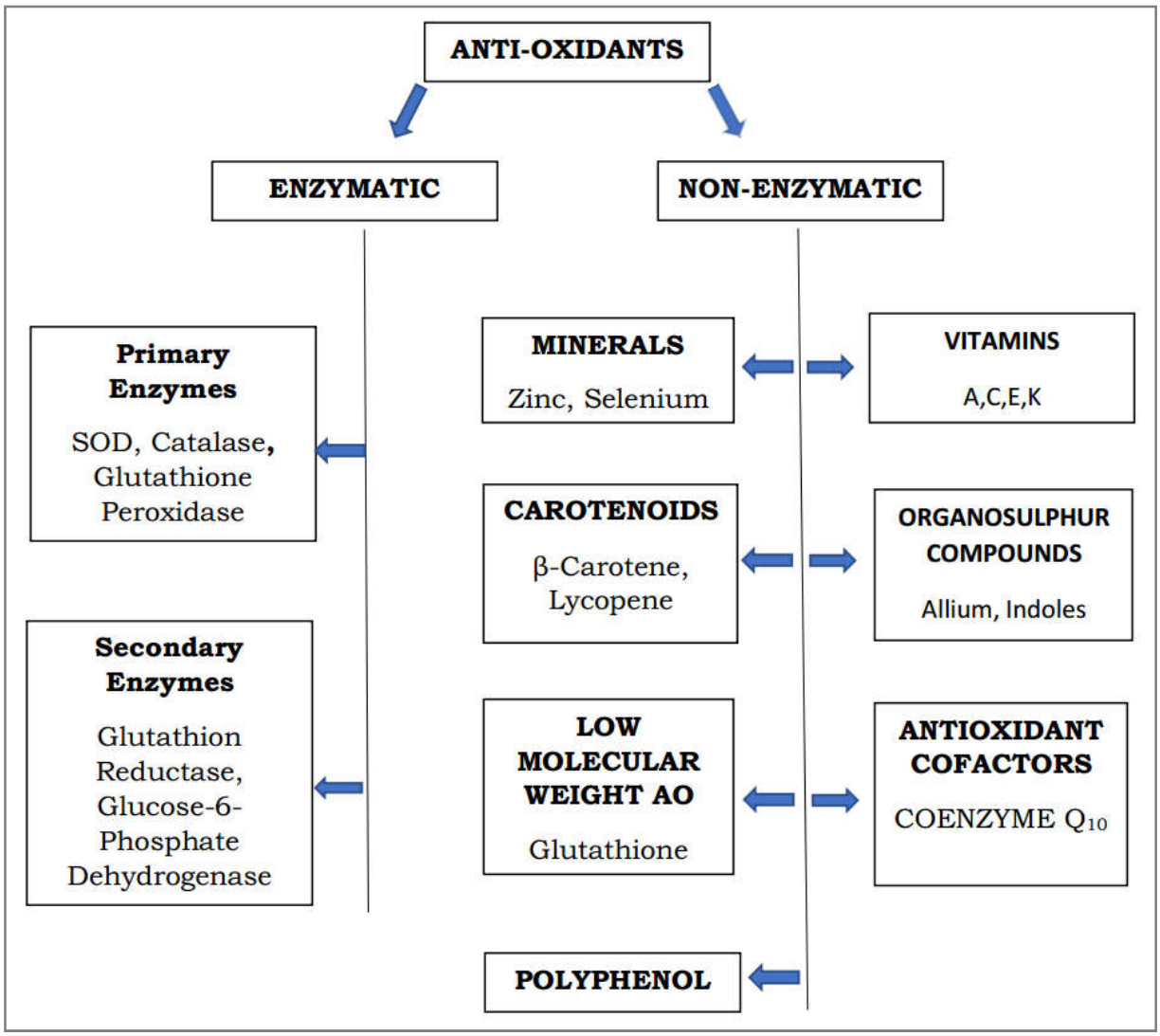
- Bleaching agents
- Composite fillings
- Dental cements
- Ceramic restoration
- Metals in restoration
- Dental implants
- Intracanal medicament

Antioxidants are defined as the substances that interact with and stabilize free radicals thereby protecting cells from the damage caused by free radicals. The equilibrium between antioxidant defense, repair systems and pro-oxidant mechanisms of cell damage may be tipped in favor of tissue destruction by increase in radical production or by alowered antioxidant defence (Vasundhara Shivanna, 2013).

Classification of Anti-Oxidants: Antioxidants can be classified into two major groups; that is, enzymatic and nonenzymatic antioxidants. Some of these antioxidants are endogenously produced, including enzymes, low-molecular-weight molecules, and enzyme cofactors. Many nonenzymatic antioxidants are obtained from dietary sources (Andrei, 2012).

The antioxidants are classified in the following manner (Andrei, 2012).

Vitamin E, flavonoids, catechins, gallic acid derivatives, salicylic acid derivatives, cinnamic acid derivatives chlorogenic acid, resveratrol, folate, curcumin, caffeine, anthocyanins and tannins are examples of polyphenolic natural antioxidants derived from plant sources (Shyam Patel, 2015). Non-phenolic secondary metabolites such as melatonin, carotenoids, retinal, thiols, jasmonic acid, eicosapentaenoic acid, ascopyrones and allicin that show excellent antioxidant activity (Finkel, 2000).



Carotenoids belong to the tetraterpene family and over 600 natural structural variants exist. Of these only about 20 are found in human plasma and tissues, including lycopene, α-carotene, β-carotene, lutein, crytoxanthine, retinol (vitamin A1) and dehydroretinol (vitamin A2) (Vasundhara Shivanna, 2013). Lycopene is one of the most potent antioxidants primarily present in tomatoes (Agarwal, 1998). Lycopene, the red pigment of tomato, is a tetra-terpene assembled from eight isoprene units composed entirely of carbon and hydrogen, containing 11 conjugated and two non-conjugated carbon–carbon double bonds (Sanjiv Agarwal, 2000). As a polyene it undergoes cis-trans isomerization induced by light, thermal energy and chemical reactions.

Lycopene from natural plant sources exists predominantly in an all-trans configuration, the most thermodynamically stable form (Nguyen, 1999). In human plasma, lycopene is present as an isomeric mixture, with 50% as cis-isomers (Clinton, 1996). Lycopene have a single-oxygen quenching ability twice as high as that of β-carotene and 10 times higher than that of α-tocopherol (DiMascio et al., 1989). It is the most predominant carotenoid in human plasma. Its level is affected by several biological and lifestyle factors. Owing to their lipophilic nature, lycopene and other carotenoids are found to concentrate in low-density and very-low-density lipoprotein fractions of the serum. Lycopene is also found to concentrate in the adrenal gland, testes, liver and prostate gland, where it is the most prominent carotenoid (Sanjiv Agarwal, 2000). It is one of the most potent antioxidants primarily present in tomatoes. It has been hypothesized to prevent carcinogenesis and atherogenesis by protecting critical cellular biomolecules, including lipids, lipoproteins, proteins, and DNA. In recent studies, serum and tissue levels of lycopene were shown to be inversely associated with the risk of breast cancer, prostate cancer, coronary heart disease, and oral premalignant lesions.[15]

Mechanism of Action: The biological activities of carotenoids such as β-carotene are related in general to their ability to form vitamin A within the body (Clinton, 1996). Since lycopene lacks the β-ionone ring structure, it cannot form vitamin A (Stahl, 1996). Its biological effects in humans have therefore been attributed to mechanisms other than vitamin A. Two major hypotheses have been proposed to explain the anticarcinogenic and antiatherogenic activities of lycopene: nonoxidative and oxidative mechanisms. The proposed mechanisms for the role of lycopene in the prevention of chronic diseases are (Sanjiv Agarwal, 2000). Among the nonoxidative mechanisms, the anticarcinogenic effects of lycopene have been suggested to be due to regulation of gap-junction communication in mouse embryo fibroblast cells. Lycopene is hypothesized to suppress carcinogen-induced phosphorylation of regulatory proteins such as p53 and Rb anti-oncogenes and stop cell division at the G_0–G_1 cell cycle phase. Astorg and colleagues proposed that lycopene-induced modulation of the liver metabolizing enzyme, cytochrome P4502E1, was the underlying mechanism of protection against carcinogen-induced preneoplastic lesions in the rat liver. Preliminary in vitro evidence also indicates that lycopene reduces cellular proliferation induced by insulin-like growth factors, which are potent mitogens, in various cancer cell lines. Regulation of intrathymic T-cell differentiation (immunomodulation) was suggested to be the mechanism for suppression of mammary tumour growth by lycopene treatments in SHN retired mice (Sanjiv Agarwal, 2000). Lycopene also has been shown to act as a hypo-cholesterolemic agent by inhibiting HMG–CoA (3-hydroxy-3-methylglutaryl– coenzyme A) reductase (Fuhramn, 1997). Lycopene has been hypothesized to prevent carcinogenesis and atherogenesis by protecting critical cellular biomolecules, including lipids, lipoproteins, proteins and DNA (Agarwal, 1998). In healthy human subjects, lycopene- or tomato free diets resulted in loss of lycopene and increased lipid oxidation (Sanjiv Agarwal, 2000), whereas dietary supplementation with lycopene for 1 week increased serum lycopene levels and reduced endogenous levels of oxidation of lipids, proteins, lipoproteins and DNA (Rao, 1998). Patients with prostate cancer were found to have low levels of lycopene and high levels of oxidation of serum lipids and proteins (Rao, 1999).

Food sources and bioavailability: Red fruits and vegetables, including tomatoes, watermelons, pink grapefruits, apricots and pink guavas, contain lycopene. Processed tomato products, such as juice, ketchup, paste, sauce and soup, all are good dietary sources of lycopene (Sanjiv Agarwal, 2000).

A lycopene from processed tomato products appears to be more bioavailable than that from raw tomatoes (Gärtner et al., 1997). The release of lycopene from the food matrix due to processing, the presence of dietary lipids and heat-induced isomerization from an all-trans to a cis conformation enhance lycopene bioavailability (Sanjiv Agarwal, 2000).

Recommended intake levels of lycopene: Estimating the daily intake of lycopene has been difficult due to the variability of reported values in the food sources. On an average, the daily intake of lycopene is estimated to be 3.7 mg. Initially, a daily intake of 25-30 mg lycopene was suggested.However,based on the results of a study where absorption of lycopene from tomato ketchup and supplement at the intake levels of 5,10 and 20 mg daily for one week were evaluated,the suggested daily intake of lycopene was modified to 5-10 mg.This level of intake can easily be achieved by ingesting several dietary sources of lycopene (Leticia, ?).

Conclusion

The current dietary recommendation to increase the consumption of fruits and vegetables rich in antioxidants has generated interest in the role of lycopene in disease prevention. However, the evidence thus far is mainly suggestive, and the underlying mechanisms are not clearly understood. Further research is critical to elucidate the role of lycopene and to formulate guidelines for healthy eating and disease prevention. Areas for further study include epidemiological investigations based on serum lycopene levels, bioavailability and effects of dietary factors, long-term dietary intervention studies, metabolism and isomerization of lycopene and their biological significance, interaction with other carotenoids and antioxidants, and mechanism of disease prevention (Sanjiv Agarwal, 2000).

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