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ANTIOXIDANT AND ITS ROLE - AN OVERVIEW

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ABSTRACT

Antioxidant plays a vital role in oral health as well as in systemic health. Some factors damage the oral tissue, leads to periodontal diseases. Medicine used for some communicable and non-communicable diseases leads to causes changes in oxidant which is cured by antioxidant. This review focuses on relationships between antioxidants and free-radical/reactive-oxygen species in the oral environment and the sources which is responsible for the antioxidant activity.

INTRODUCTION

Maintaining a good balance of oxidants and antioxidants is important for oral health as well as systemic health. Factors such as pollutants, alcohol, nicotine, hydrogen peroxide, and dental compounds and procedures can disturb the balance of oxidants in oral tissues, causing oxidative stress. Antioxidants can help to offset the imbalance. In general, antioxidants may be available through oral ingestion, diet or vitamin supplements, and in nutraceuticals. In addition, treatment of oral and dental health problems may include drug-free, natural antioxidant remedies that are available in topical oral applications such as mouth rinse, gel, paste, gum, or lozenge compositions. These topical antioxidant remedies help reduce free-radical or reactive-oxygen species, which are causative inflammatory factors in the progression of gingival and periodontal maladies [1].

ANTIOXIDANT

An antioxidantis a molecule that inhibits the oxidation of other molecules. Oxidation is a chemical reaction that transfers electrons or hydrogen from a substance to an oxidizing agent. Oxidation reactions can produce free radicals. In turn, these radicals can start chain reactions. When the chain reaction occurs in a cell, it can cause damage or death to the cell. Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions [2].

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Sources of Antioxidants

There are several thousand antioxidants, including enzymes, vitamins, minerals and other nutrients and compounds. Some antioxidants are produced within the body; others, such as vitamins A and C, must be provided by external sources. A healthy, varied diet rich in fruits and vegetables, whole grains and nuts is an excellent source of antioxidants. Antioxidants may be supplied by other external means as well [3-6].

Effect of Antioxidant

A healthy cell has a mortal enemy which is called a free radical. Free radicals constantly seek out healthy cells and attack their vulnerable outer membranes eventually causing cellular degeneration and death. The destructive effects of free radicals can be prevented with

the addition of anti-oxidants in the diet or by anti-oxidant supplements. A good anti-oxidant complex supplement actually has advantages over diet sources in that the complex has many different specific types of anti-oxidants which seek out and destroy free radicals at many various cellular sites. Patients with periodontal disease display increased PMN number and activity. This proliferation results in high degree of free radical release culminating in heightened oxidative damage to gingival tissues, periodontal ligament and alveolar bone. Damage mediated by free radicals can be mitigated by antioxidant defense system. Physiological alteration and pathological states produced by free radicals depend on disequilibrium between free radical production and antioxidant levels leading to oxidative stress [6-8].

Vitamin C - Citrus fruits and their juices, berries, dark green vegetables (spinach, asparagus, green peppers, brussel sprouts, broccoli, watercress, other greens), red and yellow peppers, tomatoes and tomato juice, pineapple, cantaloupe, mangos, papaya and guava [9].

Vitamin E - Vegetable oils such as olive, soybean, corn, cottonseed and safflower, nuts and nut butters, seeds, whole grains, wheat, wheat germ, brown rice, oatmeal, soybeans, sweet potatoes, legumes (beans, lentils, split peas) and dark leafy green vegetables [10].

Selenium- Brazil nuts, brewer's yeast, oatmeal, brown rice, chicken, eggs, dairy products, garlic, molasses, onions, salmon, seafood, tuna, wheat germ, whole grains and most vegetables [11].

BetaCarotene- Variety of dark orange, red, yellow and green vegetables and fruits such as broccoli, kale, spinach, sweet potatoes, carrots, red and yellow peppers, apricots, cantaloupe and mangos [12].

SinglePurifiedAntioxidantMolecules

Resveratrol was shown to have potential benefits in preventing or counteracting cellular damage, cancer, aging, and many other diseases. Resveratrol is a natural polyphenolicphytoalexin found in green vegetables, citrus fruit, and red grape wines. Resveratrol acts by stimulating the forkhead transcription factor family influencing the three stages of carcinogenesis: initiation, promotion, and progression [13-15]. Phloretin is a relatively potent antioxidant against peroxynitrite and lipid peroxidation. The potent activity of this molecule is due to the stabilization of its radical via tautomerisation. More recently, the combinations of topical antioxidants such as phloretin, vitamin C, and ferulic acid prevent many signs of premature aging and correct existing photo damage in skin. This antioxidant has the ability to control the level of ROS throughout skin layers. Other functional activities include its capacity to prevent the mutation that occurred in skin cells and to participate in cultures with rapid cell turnover.Tetrahydrocurcuminoids (THC) are derived from curcuminoids extracted from the roots of Curcuma longa, commonly called turmeric root [15-18].

CONCLUSION

This review identifies novel and current therapeutic protective agents in recent studies and provides some foundation to anticipate and prepare for future challenges and opportunities. The properties of each single antioxidant such as resveratrol, ferulic acid, tetrahydrocurcuminoids, and phloretin have been tested both in vitro and in vivo in order to elucidate their role and to understand their mechanism of action. Some of these single antioxidants when applied to cells possess anti-angiogenic, anti-inflammatory, antiviral, and/or anti-tumor properties. It is possible that combined antioxidant supplements will provide greater protective effects against free-radical damage to human gingival and periodontal tissues than individual antioxidants. The increasing evidence from studies of combinations of antioxidants has raised hopes that these products can be useful in the treatment of dental pathoses.

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REFERENCES

1. Figuero E, Soory M, Cerero R, Bascones A. (2006). Oxidant/antioxidant interactions of nicotine, Coenzyme Q10, Pycnogenol and phytoestrogens in oral periosteal fibroblasts and MG63 osteoblasts, *Steroids*, 71(13-14), 1062-1072.
2. Cabrera C, Artacho R, Giménez R. (2006). Beneficial effects of green tea a review. *J Am CollNutr,* 25(2), 79-99.
3. Abebe W. (2003). An overview of herbal supplement utilization with particular emphasis on possible interactions with dental drugs and oral manifestations, *J Dent Hyg,* 77(1), 37-46.
4. Carnelio S, Khan SA, Rodrigues G. (2008). Definite, probable or dubious: antioxidants trilogy in clinical dentistry. *Br Dent J*, 204(1), 29-32.
5. Letchoumy PV, Mohan KV, Stegeman JJ, et al. (2008). In vitro antioxidative potential of lactoferrin and black tea polyphenols and protective effects *in vivo* on carcinogen activation, DNA damage, proliferation, invasion, and angiogenesis during experimental oral carcinogenesis. *Oncol Res*, 17(5), 193-203.
6. Babich H, Gottesman RT, Liebling EJ, Schuck AG. (2008). Theaflavin-3-gallate and theaflavin-3-gallate, polyphenols in black tea with prooxidant properties. *Basic ClinPharmacol Toxicol*, 103(1), 66-74.

7. Houde V, Grenier D, Chandad F. (2006). Protective effects of grape seed proanthocyanidins against oxidative stress induced by lipopolysaccharides of periodontopathogens. *J Periodontol*, 77(8), 1371-1379.
8. Stoner GD, Wang LS, Zikri N, et al. (2007). Cancer prevention with freeze-dried berries and berry components. *Semin Cancer Biol*, 17(5), 403-410.
9. Mallery SR, Zwick JC, Pei P, et al. (2008). Topical application of a bioadhesive black raspberry gel modulates gene expression and reduces cyclooxygenase 2 protein in human premalignant oral lesions. *Cancer Res*, 68(12), 4945-4957.
10. Gutierrez-Venegas G, Kawasaki-Cardenas P, Arroyo-Cruz SR, Maldonado-Frias S. (2006). Luteolin inhibits lipopolysaccharide actions on human gingival fibroblasts. *Eur J Pharmacol*, 541(1-2), 95-105.
11. Kampa M, Alexaki VI, Notas G, et al. (2004). Antiproliferative and apoptotic effects of selective phenolic acids on T47D human breast cancer cells: potential mechanisms of action. *Breast Cancer Res*, 6(2), 63-74.
12. Lee YS. (2005). Role of NADPH oxidase-mediated generation of reactive oxygen species in the mechanism of apoptosis induced by phenolic acids in HepG2 human hepatoma cells. Arch Pharm Res, 28(10), 1183-1189.
13. Lesca P. (1983). Protective effects of ellagic acid and other plant phenols on benzo[a]pyrene-induced neoplasia in mice. *Carcinogenesis,* 4(12), 1651-1653.
14. Mori H, Kawabata K, Yoshimi N, et al. (1999). Chemo preventive effects of ferulic acid on oral and rice germ on large bowel carcinogenesis. *Anticancer Res*, 19(5), 1999, 3775-3778.
15. Srinivasan M, Sudheer AR, Menon VP. (2007). Ferulic Acid: therapeutic potential through its antioxidant property. *J ClinBiochemNutr*, 40(2), 92-100.
16. Balakrishnan S, Menon VP, Manoharan S. (2008). Ferulic acid inhibits 7, 12-dimethylbenz[a]anthracene-induced hamster buccal pouch carcinogenesis. *J Med Food*, 11(4), 693-700.
17. Graf E. (1992). Antioxidant potential of ferulic acid. *Free RadicBiol Med*, 13(4), 435-448.
18. Tanaka T, Kohno H, Nomura E, et al. (2003). A novel geranylated derivative, ethyl 3-(4'-geranyloxy-3'-methoxyphenyl)-2-propenoate, synthesized from ferulic acid suppresses carcinogenesis and inducible nitric oxide synthase in rat tongue. *Oncology*, 64(2), 166-175.