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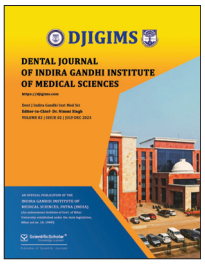

Review Article

An Understanding of Antioxidants and Oral Lesions

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ABSTRACT

The study of free radical chemistry has received a lot of attention recently. Our bodies produce free radicals, reactive oxygen and nitrogen species, and reactive nitrogen species as a result of a variety of endogenous processes, exposure to various physicochemical circumstances, or pathological conditions. For optimum physiological function, free radicals and antioxidants must coexist in balance. Oxidative stress results when the body's defenses against free radicals are overpowered. As a result, free radicals damage lipids, proteins, and Deoxyribonucleic acid (DNA) and cause a variety of human disorders. Therefore, using antioxidants from an external source can help to manage this oxidative damage. Recently, it has been suggested that synthetic antioxidants like butylated hydroxytoluene and butylated hydroxyanisole are harmful to human health. Thus, in recent years, research into natural substances with antioxidative action has been more focused.

Keywords: Oxidative stress, Free radicals, Antioxidants, and Aging

Reactive species that includes species of reactive oxygen compounds (ROS) and species of reactive nitrogen (RNS), are the results of important biological processes. Prostaglandin synthesis, purine nucleotide and arachidonic acid metabolism, prostaglandin synthesis, and enzymatic processes catalyzed by, among others, xanthine oxidase and tryptophan dioxygenase are where they are produced.[1] Numerous biological processes, such as the growth of cells, immunological defense, and sensitivity against pathogens, are influenced by reactive species. However, they might harm genetic material.[2] Antioxidants can reduce the harmful effects of overly generated reactive molecules under biological conditions.[3] These include the enzymes that repair oxidative damage, methionine sulfoxide reductase, and molecules that scavenge reactive species, such as superoxide dismutase and catalase.[4] The human body typically produces these molecules as biochemical waste materials and are then actively removed.[5]

A group of unpaired electrons within a molecule constitutes a high level of reactivity.[5] The antioxidant defense system perfectly balances producing and eradicating these reactive free radicals. To combat the impacts of harmful radicals, each cell in the human body possesses essential innate antioxidant defenses. The oral cavity corresponds to many bacteria that encounter food, drink, drugs, alcohol, nicotine, and other external chemicals. Since a disruption of the equilibrium between the two substances may cause the disease to proceed, care must be taken to maintain this balance.[6]

Any molecular entity that has an electron with no pairing in an atomic orbital and is capable of existing independently is referred to as a radical that is free of charge.[7] Oxygen-containing radicals, such as the hydroxyl radical, superoxide anion radical, peroxide of hydrogen, oxygen

singlet, hypochlorite, nitric oxide (NO) radical, and peroxynitrite radical, are most significant in numerous health situations. Volatile compounds like these can harm biologically important components like genetic material.[8] Cells are damaged, and equilibrium is upset when free radicals attack crucial macromolecules.[9] Diseases caused by harmful radicals are illustrated in [Table 1].

PRODUCTION OF UNSTABLE RADICALS

Both enzymatic and non-enzymatic mechanisms result in cells' continual production of unstable radicals. These can be produced by biochemical procedures such as the respiratory chain, destroying tissue, creating prostaglandin and the cytochrome P-450 system.[10]

Free radicals can originate from numerous internal sources, example:[11]

- Mitochondria
- Inflammation
- Phagocytosis
- Physical activity
- Ischemia/reperfusion injury
- External sources are:
- Smoking
- Contaminants from atmosphere
- Irradiation
- Solvents used in manufacturing
- Ozone

Table 1: Diseases caused by free radicals.

Organs	Diseases
1) Gastro-intestinal tract	Hepatitis
	Endotoxin liver injury
	Ulcerative colitis
	Ischemic Bowel
2) Teeth	Periodontitis
3) Vessels	Vasospasms
	Atherosclerosis
4) Multiorgan failure	Radiation
	Aging
	Cancer
	Inflammatory immune injury
	Ischemia-reperfusion injury
	Diabetes
5) Brain	Trauma
	Epilepsy
	Alzheimer's
	Parkinson's
	Dementia
6) Lung	Asthma
	Acute respiratory distress syndrome (ARDS)
	Cystic fibrosis
7) Joints	Rheumatoid arthritis
8) Heart	Heart attack
9) Skin	Sunburn
	Dermatitis
	Psoriasis
	Scleroderma
10)Kidney	Renal graft
	Glomerulonephritis
11)Eye	Retinal damage
	Macular degeneration

These radicals are produced in the following ways.

1. The electron transport mechanism in mitochondria consumes around 90% of the cell's oxygen during normal aerobic metabolism.
2. One of the processes utilized to eradicate bacteria and viruses and denature foreign proteins, or antigens, is oxidative burst produced by phagocytes, or white blood cells.
3. Xenobiotic metabolism, commonly known as detoxification of dangerous chemicals.

Therefore, factors like intense physical activity, which quickens the metabolism of cells, prolonged inflammation, sickness, having a "leaky gut" condition, getting subjected to medications, and toxic substances like smoking, emissions, and insecticides may all increase the body's oxidant load.[12]

THE BIOLOGY OF NO-CHARGE RADICAL

As we age, reactive oxygen species cause sluggish, unfavorable alterations to spread throughout the body. All people regularly go through these "normal" aging changes. Significant disorders include cardiovascular and carcinoma, two leading causes of death. Internal radical processes, which are comparable to those caused by ionizing radiation, may be the cause of tumor formation. A higher level of lipid peroxidation may cause a highly substantial association between dietary fat and oil intake and death rates from leukaemia.[13] Research on atherosclerosis suggests that the disorder may be caused by dietary lipid free radical interactions in serum and arterial wall that result in peroxides and other compounds.[14]

PERSPECTIVE ON OXIDATIVE DAMAGE

The word describes oxidative damage when radical production and antioxidant defense activity are out of balance.[15] Numerous chemical species are damaged by oxidative damage, which develops when the creation of harmful radicals and antioxidant defenses are out of balance.[16] Immediate oxidative stress may occur in cells that have been damaged. Unbound radicals have been linked to the development and

progression of dementia, Parkinson's disease, age-associated eye disease, and brain diseases.[17]

OXIDATIVE STRESS IN THE ORAL CAVITY

The issues could be internal, like ongoing inflammation, or external, such as cigarettes and pollution of the air with included chemicals. Superoxide, hydrogen peroxide, and hydroxyl radicals are examples of reactive species that are typical consequences of metabolic activity. The immune system also creates these reactive oxygen species when tissue is inflamed or injured, such as in persistent periodontal issues.[18] Antioxidants are receiving a lot of attention on a global scale. The discussion of ANTIOXIDANTS is expanded to include systemic & premalignant oral mucosal lesions & diseases, as well as oral cancer.[19]

Numerous disorders, including atherosclerosis, certain cancers, and advancing age, have been linked to oxidative damage. This damage is now believed to significantly impact all inflammatory diseases, including osteoarthritis, vascular inflammation, kidney disease, lupus erythematosus, adult pulmonary syndrome, lung disease, and elevated blood pressure.[20]

ANTIOXIDANTS

It is a sufficiently stable component to provide an electron to a runaway liberated radical, neutralizing it and lowering the radical's potential for harm.[21] Because of their small molecular weight, these antioxidants may safely react with damaging free radicals to halt the chain reaction before damaging crucial components. Several antioxidants like glutathione, ubiquinol, and uric acid—are created by the body as part of normal metabolic processes[22] beta-carotene, citrus fruits, and retinol are the principal vitamins, even though the body has a variety of complex enzyme systems that absorb harmful radicals.[23]

MECHANISM OF ACTION

Antioxidants provide a sophisticated ecosystem where harmful radicals are scavengers. Following incredibly plausible mechanisms are used by antioxidants to protect cells from free radicals.[24]

1. **Chain breaking mechanism**
   - ✓ One way to break the oxidation chain is by scavenging free radicals. E.g. albumin, bilirubin, ubiquinol, and vitamins A, C, and E.
2. **Preventive mechanism**
   - ✓ An antioxidant breaks down the free radical or chelates the transition metal. Citric acid, transferrin, ceruloplasmin, haptoglobin, and lactoferrin are a few examples.
3. **Synergistic mechanism**
   - ✓ The potency of two antioxidants is increased when combined. E.g. By mixing tocopherol, which may break chains, and citric acid, which can chelate metals.

TYPES OF ANTIOXIDANTS

Antioxidants play several defense-related roles, such as prevention, reactive scavenging, maintenance, new synthesis, the final line of protection, or adaptability. Preventative antioxidants that halt free radicals' production are the *initial line of defence*. One of the main contributors to the production of radicals in vivo, must be the metal-induced disintegration of peroxides. Some antioxidants inhibit these reactions by converting hydroperoxides and hydrogen peroxides into ethanol and water without producing harmful radicals, while certain proteins attach metallic ions to stop these reactions.

Antioxidants that scavenge the active radicals to halt or impede chain-start reactions, are the *subsequent stage of protection*. Free radicals are neutralized by various endogenous antioxidants, some of which are hydrophilic and others lipophilic. Vitamin C, uric acid, bilirubin, albumin, and thiols are hydrophilic ROS, whereas vitamin E and ubiquinol are lipophilic radicals. Tocopherol is one of the best lipophilic antioxidants for scavenging free radicals.

Maintenance and new-generation antioxidants are the *last line of protection*. Proteinases, proteases, and peptidases are proteolytic enzymes that recognise, degrade, and eliminate molecules undergoing oxidative modification. They are found in the cytosol and mitochondria of mammalian cells.

Genetic material activities are integral to the system's defense against oxidative damage. Nucleases and glycosylases are just two of the many enzymes that can repair DNA damage.[25]

CLASSIFICATION

A robust immune system and a balanced diet are necessary for the body to maintain its antioxidant state. They are divided into groups based on how easily the body absorbs them. Endogenous antioxidants are a class of compounds that have the power to scavenge harmful radicals. Exogenous antioxidants include a subclass of antioxidants that must be ingested by the body to protect cells against oxidants and support a healthy metabolism.[24]

External antioxidants: Carotenoids (beta-carotene, lycopene, lutein, zeaxanthin) Flavonoids (Quercetin, chrysin, catechin, cyanidin, genistein) Phenolic acids (Garlic acid, ellagic acid, ferulic acid, p-coumaric acid) Vitamins (A, B, C, E, K) Minerals (Selenium, iron, magnesium, copper, zinc).

Internal antioxidants: Thiols (Uric acid, Nicotinamide adenine dinucleotide phosphate hydrogen (NADPH) and

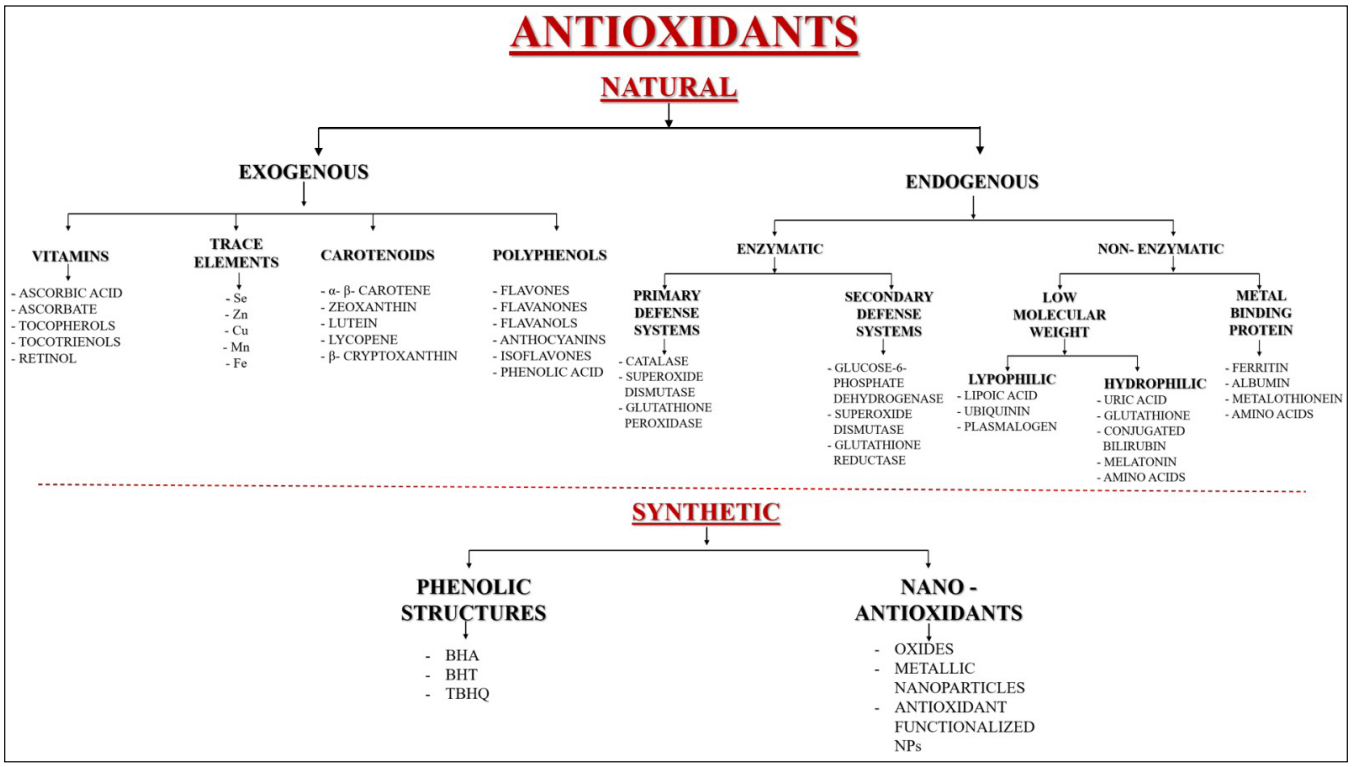

Figure 1: Classification - BHA: Butylated hydroxyanisole, BHT: Butylated hydroxytoluene, TBHQ: Tert-butylated hydroquinone, NPs: Nanoparticles

Nicotinamide adenine dinucleotide hydrogen (NADH), Bilirubin) Metal binding proteins (Albumin, Ceruloplasmin, Transferrin, Ferritin, Myoglobin) Hormones (Melatonin) Others (Glutathione, N-acetyl cysteine, Alpha lipoic acid) [Figure 1].

ANTIOXIDANTS AND CANCER PREVENTION

Interactions of harmful radicals with genetic material results in Deoxyribonucleic acid (DNA) protein cross-links, strand breaks, and base modifications. ROS damages DNA. Radicals are a significant factor in DNA chain reaction, along with double bond pyrimidine bases and the elimination of hydrogen from sugar moiety.[9] The capabilities of antioxidant B-carotene may protect against cancer due to the potential for genetic harm brought on by oxidative products. Therefore, the benefits of B-carotene photoprotective properties may protect against Ultraviolet (UV) light-induced cancer. B-carotene may also have an anticarcinogenic effect by altering how the liver processes carcinogens.[26] Vitamin C may help prevent cancer.[27] Some proposed methods by which vitamin C may affect carcinogenesis include antioxidant actions, decreasing the production of nitrosamines, and increasing the immune response.[28] The most important etiology of mouth cancer is tobacco use. Long-term tobacco smoking harms the body's oxidative defenses. Alcoholics also generate an excessive amount of free radicals. Single nucleotide polymorphism in Cytochrome P450 2E1 (CYP2E1) brought on by prolonged ethanol usage can increase ROS generation.[29] Combining alcohol and tobacco usage increases the likelihood that dysplastic disorders may progress to oral cancer.[30] Antioxidant micronutrients block transforming growth factor (TGF) alpha that stops angiogenesis in cancer.[31] Antioxidants are thought to be one of the chemopreventive medications. Retinoids, beta-carotene, curcumin, lycopene, zinc, selenium, vitamin C (ascorbic acid), vitamin E (tocopherol), and vitamin A (carotene) are known to be protective against mouth cancer. Retinoids and curcumin have been found to have different anti-mutagenic and anti-proliferative effects.[31]

Treatment: Vitamin A (1 mg/Kg/day), Beta carotene (25,000-100,000 IU/day), Selenium (40-75 microgram/day), Vitamin E (400-800 mg/day), Vitamin C (140-500 mg/day), Curcumin (300 mg/day), Lycopene (12 mg/day).

NUTRITION WITH FUNCTIONALITY AS A VISION

Functional eating is evolving from its historical focus on maintaining health to include the exciting potential of nutrition to promote health and ward off chronic disorders. Foods that offer consumers additional physiological benefits and essential nourishment are called "Nutritional foods."[32]

PRO-OXIDANT EFFECTS OF ANTIOXIDANTS

Antioxidants are well-known to act as pro-oxidants in foods and in vitro when given in high doses and under specific circumstances (i.e., by encouraging the production of oxygen free radicals). Ascorbate can occasionally worsen DNA damage in people, according to current studies. The recently discovered "redox signaling" of antioxidants is strongly related to their paradoxical activity (pro-oxidant impact). Whether antioxidants from food sources, particularly those in high concentrations, have pro-oxidant effects on people is still debated among scientists.[33]

OXIDANTS IN ORAL LESIONS OF THE MOUTH

Numerous investigations are undertaken to ascertain the importance of oxidants in the therapy for mouth issues, including testing facilities, experimental animals, statistical surveys, and therapeutic trials. The following are some antioxidants' potential therapeutic applications for oral mucosal ulcers.[34]

1. Preventing the growth of abnormalities in people at high risk who have visually normal-looking mucosa and absence of either oral Potentially Malignant Disorders (PMDs) or cancerous abnormalities.
2. Management of problems that could be malignant
3. To prevent recurrence of previously treated abnormalities.

Oral lichen planus (OLP) and lichenoid reactions: keratinocyte antigen presentation changes due to numerous cytokines produced by T cell lymphocytes. This inflammatory process kills the basal epithelium's cells.[35] Malondialdehyde (MDA) and 4-hydroxy-2-nonenal, two indicators of oxidative stress, are present in greater concentrations in 35 OLP patients. They are byproducts of lipid peroxidation. Antioxidants may be administered to OLP patients to minimize the condition's propensity to change. The most potent biological antioxidant is lycopene, a well-known carotenoid with a peculiar ability to bind to chemical species that react to oxygen. In a placebo-controlled study, giving OLP patients supplements containing 4–8 mg/day of lycopene for three months had beneficial results; the burning feeling was reduced by 84%, and oxidative stress was reduced by 67% compared to the control group.[36] Curcuminoids, found in turmeric, have anti-inflammatory and antioxidant properties. Greater doses of curcumin (up to 6,000 mg/day), according to trials to date, dramatically improved symptom control in a large percentage of OLP patients.[37]

Treatment: Lycopene (6–60 mg/day), Curcumin (6000 mg/day), Selenium (40–80 microgram/day).

Oral Leukoplakia (OL): The most common ailment, it has a complicated etiology and may be cancerous. Lycopene can be used to treat leukoplakia successfully. Lycopene promotes the production of the gene-generating connexion-43, a gap junction protein, independently of its provitamin-A or antioxidant actions.[38] Isotretinoin is an effective treatment for oral leukoplakia. Topical 13-retinoic acid (0.1 percent isotretinoin gel) therapy resulted in a complete clinical response, and none of the patients reported any harmful effects. Taking 3–9 lakh International Unit (IU) of vitamin A daily helped to cure leukoplakia partially or totally.[39]

Treatment: Beta-carotene (20–90 mg/day) and systemic dose (300,000 IU), Retinoids vitamin A/Retinol (0.5–1 mg/day), Lycopene (8 mg/day), Vitamin E (8–10 mg/day), Curcumin (4000–8000 mg/day).

Oral submucous fibrosis: Areca nuts are among the most addictive foods on the planet because they contain alkaloid arecoline, polyphenols, and trace metals like copper. The first study to demonstrate that areca nut aqueous extracts can generate superoxide anion and hydrogen peroxide at pH levels higher than 9.5 was conducted by Nair et al.[40] Lycopene has been seen to significantly reduce clinical signs and symptoms at a dosage of 16 mg per day when used in conjunction with other therapy methods including intralesional injection.[41] Other synthetic antioxidants have been found to possess potent antioxidant, DNA-protective, and antimutagenic effects, such as curcuminoids.[42]

Treatment: Lycopene (16 mg/day), Curcumin (500-800 mg/day), Alpha lipoic acid (50 mg/day), Alpha tocopheryl acetate (10 IU).

Aphthous stomatitis: is a disorder that results in the development of oral mucosa of painful circular ulcers with clearly defined reddened edges and a yellowish central area. Full healing occurred in 35% of patients between 2 and 4 days, 90% 7 days, and all patients treated with quercetin in >7 days when quercetin was given topically to treat minor mouth ulcers.[43] Other herbal topical therapies, like curcumin, contain anti-ulcerogenic properties.[44]

Treatment: Quercetin (500–1000 mg/day), Curcumin (150 mg/day).

CONCLUSION

Our bodies produce reactive substances such as nitrogen and oxygen due to various internal systems' responses to different physicochemical circumstances or diseases. For optimum functioning of the body, antioxidants and free radicals must coexist in balance. Oxidative damage results when the body's defenses against radicals that are free are overpowered. As a result, reactive oxygen species damage lipids, proteins, and DNA and cause various human disorders. Therefore, using external oxidants can help manage this oxidative damage.

Recently, it has been suggested that synthetic antioxidants like butylated hydroxytoluene and butylated hydroxyanisole are harmful to the well-being of humans. As a result, efforts to find potent, safe, natural substances with antioxidative action have increased in recent years.

Declaration of patient consent

Patient's consent is not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

Use of Artificial Intelligence (AI)-Assisted Technology for manuscript preparation

The authors confirm that there was no use of Artificial Intelligence (AI)-Assisted Technology for assisting in the writing or editing of the manuscript and no images were manipulated using the AI.

REFERENCES

1. Halliwell B, Gutteridge C. Free radicals in biology and medicine. 5th ed. Oxford, UK: Oxford University Press; 2015. p. 707
2. Schieber M, Chandel N. ROS Function in Redox signaling and oxidative stress. Curr Biol 2014;24:453–62.
3. Poljsak B, Šuput D, Milisav I. Achieving the balance between ROS and antioxidants: When to use the synthetic antioxidants. Oxid Med Cell Longev 2013;2013:956792.
4. Weissbach H, Resnick L, Brot N. Methionine sulfoxide reductases: History and cellular role in protecting against oxidative damage. Biochim Biophys Acta Proteins Proteom 2005;1703:203–12.
5. Rahman K. Studies on free radicals, antioxidants, and co-factors. Clin Interv Aging 2007;2:219–36.
6. Hemanthakumar S, Narmadha C, Saraswathi Gopal K, Srividhya S, Mahesh Kumar P. Antioxidants in oral mucosal diseases: A scoping remedy. Annals of RSCB [Internet] 2021;25:18549-56.
7. Cheeseman KH, Slater TF. An introduction to free radicals chemistry. Br Med Bull 1993;49:481–93.
8. Young IS, Woodside JV. Antioxidants in health and disease. J Clin Pathol 2001;54:176–86.
9. Lobo V, Patil A, Phatak A, Chandra N. Free radicals, antioxidants and functional foods: Impact on human health. Pharmacogn Rev 2010;4:118–26.
10. Liu T, Stern A, Roberts LJ. The isoprostanes: Novel prostanglandin-like products of the free radical catalysed peroxidation of arachidonic acid. J Biomed Sci 1999;6:226–35.
11. Ebadi M. Antioxidants and free radicals in health and disease: An introduction to reactive oxygen species, oxidative injury, neuronal cell death and therapy in neurodegenerative diseases. Arizona: Prominent Press; 2001. p. 13–5.
12. Sarma AD, Mallick AR, Ghosh AK. Free radicals and their role in different clinical conditions: An overview. Int J Pharm Sci Res 2010;1:185–92.
13. Lea AJ. Dietary factors associated with death rates from certain neoplasms in man. Lancet 1966;2:332–3.
14. Harman D. Role of free radicals in aging and disease. Ann N Y Acad Sci 1992;673:126–41.
15. Rock CL, Jacob RA, Bowen PE. Update o biological characteristics of the antioxidant micronutrients-Vitamin C, Vitamin E and the carotenoids. J Am Diet Assoc 1996;96: 693–702.
16. Mc Cord JM. The evolution of free radicals and oxidative stress. Am J Med 2000;108:652–9.
17. Rao AL, Bharani M, Pallavi V. Role of antioxidants and free radicals in health and disease. Adv Pharmacol Toxicol 2006;7: 29–38.
18. Sculley DV, Langley-Evans SC. Salivary antioxidants and periodontal disease status. Proc Nut Soc 2002;61:137–43.
19. Bhateja S. Role of antioxidants in oral medicine. Int J Pharm Sci Res 2012;3:1971–5.
20. Stefanis L, Burke RE, Greene LA. Apoptosis in neurodegenerative disorders. Curr Opin Neurol 1997;10:299–305.
21. Halliwell B. How to characterize an antioxidant - An update. Biochem Soc Symp 1995;61:73–101.
22. Shi HL, Noguchi N, Niki N. Comparative study on dynamics of antioxidative action of α-tocopheryl hydroquinone, ubiquinol and α- tocopherol, against lipid peroxidation. Free Radic Biol Med 1999;27:334–46.
23. Levine M, Ramsey SC, Daruwara R. Criteria and recommendation for Vitamin C intake. JAMA 1991;281:1415–23.
24. Brar SK, Dhillon GS, Soccol CR. Biotransformation of Waste Biomass into High Value Biochemicals. Book Springer New York, NY. 2014; Edition 1;XV, 504.
25. Niki E. Antioxidant defenses in eukaryotic cells. In: Poli G, Albano E, Dianzani MU, editors. Free radicals: From basic science to medicine. Basel, Switzerland: Birkhauser Verlag; 1993. p. 365–73.
26. Poppel GV, Golddbohm RA. Epidemiologic evidence for β – carotene and cancer prevention. Am J Clin Nutr 1995;62: 1393-5.
27. Glatthaar BE, Horing DH, Moser U. The role of ascorbic acid in carcinogenesis. Adv Exp Med Biol 1986;206:357–77.
28. Sokol RJ. Vitamin E deficiency and neurologic diseses. Annu Rev Nutr 1988;8:351–73.
29. Poulsen HE, Prieme H, Loft S. Role of oxidative DNA damage in cancer initiation and promotion. Eur J Cancer Prev 1998;7: 9–16
30. Bonner MY, Arbiser JL. The antioxidant paradox: What are antioxidants and how should they be used in a therapeutic context for cancer? Future Med Chem 2014;6:1413–22.
31. Saraswathi Gopal K. Chemopreventive agents in head and neck cancer. Int J Curr Res 2017;9:47228–34.
32. Devasagayam TPA, Tilak JC, Singhal R. Functional foods in India; history and scope in angiogenesis. In Losso JN, Shahidi F,

Bagchi D, editors. Functional and medicinal foods. New York: Marcel Dekker Inc.; 2001.
33. Devasagayam TP, Tilak JC, Boloor KK, Sane KS, Ghaskadbi SS, Lele RD. Free radicals and antioxidants in human health: Current status and prospects. J Assoc Physicians India 2004:52:794–804.
34. Shetti A, Keluskar V, Aggarwal A. Antioxidants: Enhancing oral and general health. J Indian Acad Oral Med Radiol 2009;21:1.
35. Boccellino M, Di Stasio D, Romano A, Petruzzi M, Lucchese A, Serpico R *et al*. L planus: Molecular pathway and clinical implications in oral disorders. J Biol Regul Homeost Agents 2018;32:135–8.
36. Saawarn N, Shashikanth M C, Saawarn S, Jirge V, Chaitanya NC. PRL in managing oral lichen planus: A placebo-controlled study. Indian J Dent Res 2011;22:639–43.
37. Singh V, Tiwari S, Das S, Gupta S, Malkunje L, Pal M. Turmeric - A new treatment option for lichen planus: A pilot study. Natl J Maxillofac Surg 2013;4:198.
38. Katakwar P, Metgud R, Naik S, Mittal R. Oxidative stress marker in oral cancer: A review. J Cancer Res Ther 2016;12:438–46.
39. Ribeiro AS, Salles PR, da Silva TA, Mesquita RA. A review of the nonsurgical treatment of oral leukoplakia. Int J Dent 2010;2010:1–10.
40. Nair U, Bartsch H, Nair J. Alert for an epidemic of oral cancer due to use of the betel quid substitutes gutkha and pan masala: A review of agents and causative mechanisms. Mutagenesis 2004;19:251–62.
41. Panneer Selvam N, Anand Dayanand A. Lycopene in managing oral submucous fibrosis. Asian J Pharm Clin Res 2013;6: 58–61.
42. Das A D, Balan A, KT S. Comparative study of the efficacy of curcumin and turmeric oil as chemopreventive agents in oral submucous fibrosis: A Clinical and Histopathological Evaluation. J Indian Acad Oral Med Radiol 2010;22:88–92.
43. Calvo TR, Lima ZP, Silva JS, Ballesteros KVR, Pellizzon CH, Hiruma-Lima CA, *et al.* Constituents and antiulcer effect of Alchornea glandulosa: Activation of cell proliferation in the gastric mucosa during the healing process. Biol Pharm Bull 2007;30:451–9.
44. Halim D, Khalik N, Taib H, Pohchi A, Hassan A, Alam M. Novel Material in the Treatment of Minor Oral Recurrent Aphthous Stomatitis. Int Med J (1994) 2013;20:392–4.

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