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*Review Article*

# OXIDATIVE STRESS AN ORAL STRESS- A REVIEW

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**A R T I C L E I N F O**

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**A B S T R A C T**

Oxidative stress is a physiological process in the human system, which is necessary for the sustainability and viability of the organism, however when this normal process is altered by any diseases process or imbalance in curbing the free radicles causes the system to react more vulnerably. This imbalance or antioxidant defect mechanism in the body due to any Aetiology factore, paves the way for more damages to the system, which finally results in a disease state, showing various clinical features .Various pathologies are directly or indirectly linked to this process. A deeper understanding is necessary to prevent such imbalance, which culminates in various systemic conditions.



## INTRODUCTION

Stress can be defined as a state of worry or mental tension caused by a difficult situation. Stress is a natural human response that prompts us to address challenges and threats in our lives. Stress affects both the mind and the body. Stress makes it hard for us to relax and can come with a range of emotions, including anxiety and irritability. Chronic stress can worsen pre-existing health problems and may increase our use of alcohol, tobacco and other substances. Stressful situations can also cause or exacerbate mental health conditions, most commonly anxiety and depression[1].

Similarly oxidative stress from different diseases can have deletedriousaffect on human system .This type of stress accumulate when there is any imbalance in normal function both of physical and mental health.

In Humans, there is a delicate equilibrium between the production and the elimination – by antioxidant defense system – of the so-called "free radicals". The breaking of this balance- named as "oxidative stress", may induce a cellular damage, with differing degrees of severity, leading ultimately, over time, to early aging and to many diseases.[2]

Oxidative stress in a pathological condition triggered by the damaging action – on the cells and tissues of the body – of abnormally increased amounts of free radicals. Oxidative stress is the direct consequence of an increased generation of free radicals and/or a reduced physiological activity of antioxidant defenses against free radicals.[3]

In physiological conditions, both enzymatic and non-enzymatic systems preserve the oxidant/antioxidant status. However these systems are overwhelmed during oxidative stress, which is a metabolic derangement due to an imbalance caused either by an excessive generation of ROS or by a diminished capacity of the antioxidant defense system. Impairment of cell function might be caused by several factors, typically more than one acting at the same time, enhancing the same pathophysiological pathway or other, resulting in various systemic diseases[4].

Oxidative stress is defined as a"state in which oxidation exceeds the antioxidant systems in the body secondary to a loss of the balance between them. It results in hazardous events such as lipid peroxidation and oxidative DNA damage, but also physiologic adaptation phenomena and regulation of intracellular signal transduction. From a clinician point of view if certain chemicals are released during this process called as 'bomarkers' , then certain diseases process kicks inn to start.[5]Followed by Oxidative stress occurs with the generation of free radicals and active intermediates.[6]

### Generation of Reactive Oxygen and Nitrogen Species

The generation of ROS is a physiological and normal to aerobic life. In mammalian, under physiological conditions, cells metabolize approximately 95% of the oxygen (O2) to water, without formation of any toxic intermediates. Several studies were put forward which agree that, in normal conditions, a minimal 5% of O2 is metabolized through univalent reduction, following four different reactions or stages:

Reaction 1: $O2 + e \rightarrow O2^-$
Reaction 2: $O2^- + e \rightarrow H2O2$
Reaction 3: $H2O2 + e \rightarrow \bullet OH$
Reaction 4: $\bullet OH + e \rightarrow H2O$

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Through these four reactions three highly toxic species are formed, two of them being free radicals: O2•– and hydroxyl radical (•OH). Hydrogen peroxide (H2O2) is still a highly reactive compound. This four reactions explains in general terms the mitochondrial generation of ROS in normal cellular metabolism.[7]

Free radicals are single or grouped atoms having at least one external orbital "occupied" byone single electron ("unpaired") instead of a couple of electrons ("lone pair").Antioxidants are chemical or biological agents able to neutralize the potentially damaging action of free radicals.[8]

An imbalance between production and accumulation of oxygen reactive species (ROS).in cells and tissues and the ability of a biological system to detoxify these reactive products give way different disease to kick start[9].

The current accepted system of oxidative stress should also add with yet another system wherein nitrogen also plays crucial role in the metabolic system. Reactive oxygen intermediate (ROI) and reactive nitrogen intermediate (RNI) are constantly produced under physiological conditions and is a crucial event in living organisms.[10]

During the metabolic processes, these radicals act as mediators for the transfer of electrons in various biochemical reactions. The continuous production of free radicals during the metabolic processes culminated in the development of antioxidant defense mechanisms. [11]This system may be overwhelmed by various pathological or environmental factors so that a fraction of ROS may escape destruction and form the far more reactive hydroxyl radical.[10]

This ubiquitous antioxidant defense system, consists of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase and low molecular-weight antioxidants such as ascorbate, α-tocopherol and glutathione, cys- teine, thioredoxin, vitamins. These are intended to limit the intracellular levels of these reactive species and control the occurrence of damage caused by them.[12,8,6,11]

The various metabolic process like protein phosphorylation, activation of several transcriptional factors, apoptosis, immunity, and differentiation, are all dependent on a proper ROS production and presence inside cells that need to be kept at a low level. When ROS production increases, they start showing harmful effects on important cellular structures like proteins, lipids, and nucleic acids.[6]

The structural modifications in the molecules of nucleic acids, proteins and lipids caused by increased concentration of reactive oxygen species (ROS) and/or reactive nitrogen species (RNS) lead to various metabolic changes that may contribute to the development of neurological diseases, cardiovascular diseases, cancer, type 2 diabetes, cancer and aging, heart failure, hypertension, preeclampsia and atherosclerosis, among others.[6,8,11]

**Major Active Oxygen Species**

O2 Superoxide radical , H2O2 Hydrogen peroxide, HO Hydroxyl radicalO2 Singlet oxygen , HOO Hydroperoxyl radical, LOOH Alkyl hydroperoxide LOOA lkylperoxyl radical, LO Alkoxyl radical, CIO Hypochlorite ionFe4OFerryl ion, Fe5OPeriferryl ion, NO Nitric oxide.[6]

**Biomarkers of Oxidative Stress**

The biomarkers that can be used to assess oxidative stress have been attracting interest because the accurate assessment of such stress is necessary for investigation of various pathological conditions, as well as to evaluate the efficacy of drugs. The body fluids like saliva, blood, urine, and other biological fluids may provide information of diagnostic value. Various free radicle produced in certain process are:-

1. Lipid peroxides malondialdehyde, and 4-hydroxynonenal as markers for oxidative damage to lipids.
2. Isoprostan as a product of the free radical oxidation of arachidonic acid.
3. 8-oxoguanine (8-hydroxyguanine) and thymineglycol as indicators of oxidative damage to DNA.
4. carbonyl protein, hydroxyleucine, hydrovaline, and nitrotyrosine oxidation of protein and amino acids.[6]

**Antioxidant defense system**

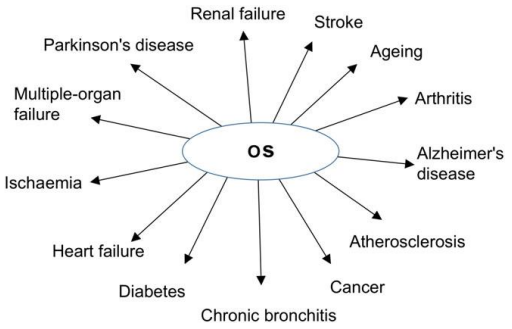
The various enzymatic defense system act through mechanisms of preventing and/or controlling the formation of free radicals and species involved with the initiation of chain reactions that culminate in propagation and process of occurrence of oxidative damage. The first line of the antioxidant defense to exogenous toxins includes the enzymes involved in phase I and II metabolism. The phase I metabolism is responsible for increased compound polarity through oxidation, reduction or hydrolysis reactions. The phase II metabolism, in the other hand, is responsible for facilitating the cellular export of those compounds; its reactions are mainly glucuronidation, acetylation and sulfation. The enzymes that compose the cytochrome P450 are the most responsible for oxidation of drugs, chemicals and various endogenous substrates, such as eicosanoids, cholesterol, vitamin D3 and arachidonic acid,.[8]

**Causes responsible for a reduced antioxidant defense**

In healthy condition the body is able to prevent free radicals accumulation. A reduced effectiveness of a system is substantially attributed to an absolute or relative deficiency of antioxidants, independently of the involved mechanism. The various factors responsible for this reduced defence are attributed to the following: Hypovitaminosis, monotonous diet, Malabsorption syndromes, celiac disease, Uptake and/or carrier deficiency, Genetic and/or iatrogenic factors.[2]

**The classical oxidative stress theory of disease.[13,5]**

The concept behind the OS theory of disease is that the metabolism of molecular oxygen (O2) by the cell results in the production of ROS, including hydrogen peroxide (H2O2), the hydroxyl radical (OH•) and the superoxide radical (O2 all of which can be toxic by reacting with cellular macromolecules. In 1956, Harman, postulated the 'free radical theory of ageing' hypothesizing that the degenerative process of ageing has a free radical mechanism in common with cancer and radiation toxicity.




A typical scheme generalizing the OS theory of disease

**Oxidative stress and theory of aging**

Aging is the progressive loss of tissue and organ function over time. The free radical theory of aging, later termed as oxidative stress theory of aging, is based on the structural damage-based hypothesis that age-associated functional losses are due to the accumulation of oxidative damage to macromolecules (lipids, DNA, and proteins) by RONS. However the exact mechanism of this is not clear. Oxidative stress, cellular senescence, and consequently, SASP factors are involved in several acute and chronic pathological processes, such as CVDs, acute and chronic kidney disease (CKD), neurodegenerative diseases (NDs), macular degeneration (MD), biliary diseases, and cancer.[14,15]

**Clinical implications of oxidative stress[16,17]**

The oxidative stress, being a merely biochemical condition, generally doesn't exhibit any specific clinical symptoms nor clinical signs. Therefore, it will remain unknown, with unavoidable damage to the patient, until the clinician suspects its existence and decides to perform specific biochemical tests, i. e. the d-ROMs test and the BAP test.

Oxidative stress plays important role in the pathophysiology of various diseases. Cellular antioxidants are known to change their redox state and they can be targeted for destruction, regulate oxidative processes involved signal transduction, effect gene expression and the pathways of cell proliferation and death. Impaired endogenous antioxidant system results in accumulation of free radicals. [18]

Emphasis has been placed on the role of oxidative stress inchronic diseases, such as Alzheimer's disease and atherosclerosis. Hence evaluating the oxidative stress rely on detecting levels of in-dividual by products of oxidative damage or by determining the to tallevelsor activity of individual antioxidant enzymes.[19, 20]

Ageing and chronic degenerative pathologies demonstrate the shared characteristics of high bioavailability of reactive oxygen species (ROS) and oxidative stress, chronic/persistent inflammation, glycation, and mitochondrial abnormalities. The deleterious repercussions of immoderate reactive oxygen and nitrogen species (RONS) is critical and may curb the progression of ageing and chronic degenerative syndromes, such as chronic degenerative pathologies, including neurodegenerative diseases, such as Alzheimer's disease (AD) and Parkinson's disease (PD), cardiovascular diseases CVD, diabetes mellitus (DM), and chronic kidney disease .[21]

Increased steady-state ROS levels can be promoted by drug metabolism, over expression of ROS-producing enzymes, or ionizing radiation, as well as due to deficiency of antioxidant enzymes.[22, 23]

**Oxidative stress and molecular damage**

The cellular process of oxidative stress arises from an imbalance between oxidants and antioxidants in favor of excessive generation of free radicals or removal speed thereof. This process leads to the oxidation of biomolecules with consequent loss of its biological functions and/or homeostatic imbalances, whose manifestation is the potential oxidative damage to cells and tissues. Accumulation of ROS/RNS can result in a number of deleterious effects such aslipid peroxidation, protein oxidation and DNA damage.

**DNA Damage**

DNA and RNA are chemically unstable and vulnerable to hydrolysis, nonenzymatic methylation and oxidation, due to its susceptibility to endogenous and exogenous damage. Endogenous genotoxic agents are mainly produced by cellular metabolism and composed of ROS and RNS, estrogen metabolites and aldehydes produced by lipid peroxidation.

This is considered as the important one as ROS-induced cellular modifications- as DNA is not synthesized de novo but copied, perpetuating by this way those modifications, and hence inducing mutations and genetic instability. Oxidative DNA modifications have been suggested as important contributory factors to the mechanism in carcinogenesis, diabetes and natural aging. The main responsible ROS of DNA damage is •OH, which reacts with all components of the DNA molecule, damaging both purine and pyrimidine bases and the deoxyribosebackbone.RNS such as ONOO- and •NO have also been implicated in DNA damage.

**Protein damage**

The effects of oxidation in proteins can be observed in impaired protein folding, side-chainoxidation and backbone fragmentation, resulting in loss of function and stop a variety of biochemical processes[6].

**Oxidative stress and infection**

The pathological effects of NO and O2 − in virus infection are in clear contrast to their beneficial antimicrobial effects in bacterial and fungal infections. In virus infections, NO and ONOO−, which are primitive host-defense molecules, cause nonspecific oxidative damage in virus infected tissue, leading to various pathological events. Virus-induced oxidative stress has been reported during HIV, influenza virus, HBV, hepatitis C virus, encephalomyocarditis virus (EMCV), respiratory syncytial virus (RSV), dengue virus (DENV) and others. [6]

**Temporomandibular (TMB) Joint Disorders**

Mechanical stresses can lead to ROS-induced oxidative stress of the temporomandibular joint (TMJ),results in tissue damage, which further propagates to temporomandibular disorder(TMD).Using electron spin resonance (ESR) spin trapping technique, free radicles are found in study models of rat or in synovial fluid (SF) of the TMD patient.

ROS can be generated in the TMJ by several pathways: they include

1) direct mechanical in-jury
2) hypoxia-reperfusion and
3) arachidonic acid catabolism to the articular tissues.

The ROS, especially HO• is responsible for lipid peroxidation and disruption of cellular homeostasis.Various free radicles can affect the normal tmj function which include:.

1) Reduction of SF viscosity by depolymerization and/or molecular configuration of hyaluronic acid (HA)
2) Re-duction of lubrication of the articular surfaceby deterioration of the surfaceactive phospholipid (SAPL) layer, which acts as an extremely efficient boundary lubricant and protector of articular surfaces.
3) Breakdown of collagen proteoglycans.
4) Activation of cartilage degrading enzymes such as matrix metalloproteinases.

Free radicals which are produced in various process in the joint, may result in a deterioration, lubrication of the articular surface, thus further proceed- ing to the internal derangement (ID) of the TMJ. Various literatures show that free radicals may cause molecular deterioration, which further proceeds to degenerative changes in the TMJ.

**Rheumatoid Arthritis**

Rheumatoid arthritis is a chronic inflammatory disorder affecting the joints and surrounding tissues, characterized by macrophages and activated T cell infiltration. An increase in reactive oxygen species (ROS) plays an important role in the pathogenesis of rheumatoid arthritis. In RA, ROS are important intracellular signaling molecules in the cells of the immune system that amplify the synovial inflammatory-proliferative response. T-cells are exposed to increased oxidative stress and be- come refractory to growth and death stimuli, which further contributes to the perpetuation of the immune response. Persistent inflammation results in destruction of cartilage and bone. This occurs through number of mechanisms, including oxidative and proteolytic breakdown of collagen and proteoglycans. Once sequestered within the joint space, neutrophils degranulate and re- lease a variety of potentially harmful enzymes and pep- tides.

Oral diseases such as dental caries, lichen planus, oral cancer, and most importantly chronic periodontitis are also believed to be linked to oxidative stress. Data from the literature report a direct link between free radical levels, oxidative stress and inflammatory states. This association is also found in the most common inflammatory diseases and potential neoplastic lesions of the oral cavity diseases.[24,25]

Oxidative stress in oral disease is related to other systemic diseases in the body such as periodontitis, cardiovascular, pancreatic, gastric, and liver diseases. Among all oral diseases, the periodontal disease (comprising gingivitis and periodontitis), accounted in a major portion of the oral diseases. Oxidative stress was involved in the progression of periodontitis, a chronic inflammatory disease of the periodontal tissue, caused by disturbance in the regulation of the host inflammatory in response to bacterial infection.[26]

There is increasing evidence linking periodontitis to systemic diseases such as metabolic syndrome (MetS), diabetes and, especially, CVD.WHICH ARE CAUSED due to the high OS accumulation and these diseases are indirectly related to the various oral lesions.[27]

Diabetes and periodontitis. Periodontal disease has been recognized as the "sixth complication of both types of diabetes". Many biochemical pathstrictly associated with hyperglycemia such as glucose auto-oxidation, polyol pathway, prostanoid synthesis and protein glycation can increase the production of ROS.[27]

The Role of Oxidative Stress: The fundamental causative factor is the engagement of host and the bacterial enzymes in the destroying the periodontium. PMNs arrive at the site. There is release of ROS and proteolytic enzymes catalyzed by the NADPH oxidase. Rapid release of oxygen from the PMN by the mechanism called the "respiratory burst..ROS are also produced byosteoclasts in the bone and might have influence bone resorption. PMNs thereby lead to ROS formation resulting in the destruction of periodontal tissues.[28.29]

**Recurrent aphthous stomatitis .[30]**

The most common ulcerative disorder of the oral mucosa, RAS is characterized by painful single or multiple round shallow ulcers with well demarcated erythematous margin and yellowish-greyish pseudomembranous central area. The ulcers last from 7 to 14 days and reappear at intervals of a few months to a few days. The etiology of RAS lesions is not entirely clear, but severallocal (trauma) systemic, genetic, immunological , nutritional , allergic and microbial , factors have been proposed as causative agents. All these issues can perturb the oxidant-antioxidant compromising the immune system. In a recent study, conducted by Ziaudeen and Ravindran, in RAS cases, they found an increase in mean salivary SOD and a reduction of the activity of GPx and UA in the study group compared to the controls equilibrium of the organism thus triggering the formation of free radicals. They also found that infiltration of immune cells into the lesion led to an increase in free radical concentration.DNA damage was observed to be significantly higher than in the control group.[30]

Oral lichen planus (OLP). A common chronic inflammatory disease of the oral mucosa whose etiologic basis is not yet fully understood, OLP is manifested as an exaggerated response to the body's immune system. The most common is the reticular form, which is characterized by white keratotic dots and lines, called Wickham's striae, surrounded by anerythematous area, reflecting sub epithelial inflammation. Most lesions are bilateral and located on the buccal mucosa, on the tongue, in the vestibule and on the gingivae. Patients with OLP have highlevels of lipid peroxidation products such as malondialdehyde (MDA) and 4-hydroxy-2-nonenalmarkers trigger a sequence of biological responses (including apoptosis initiation) by influencing the levels of B-cell lymphoma 2 and BCL2-associated X proteins, the recruitment of pro-inflammatory T-lymphocytes and nuclear localization and activity of factor kappa B.[31]

Oral pemphigus vulgaris (OPV). Pemphigus vulgaris is an autoimmune disease involving both the skin and mucosal areas, in which acantholysis (the loss of cell adhesion) causes intraepithelial blister. Pemphigus is typically characterized byimpairment of the desmosomes by IgG-antibodies, against the extracellular domains of desmogleins with intraepithelial immune deposits. the highest activity of antioxidant enzymes (SOD, catalase and GPx) and a reduced total antioxidant capacity in patients with OPV.[32]

**Oral leukoplakia.**

Oral leukoplakia is the most common potentially malignant disorder of the oral mucosa. Many cases of oral cancer are preceded by a variety of potentially malignant oral disorders,

of which leukoplakia appears to be most common. Changes in saliva composition, caused by pathological processes as oral potentially malignant lesions, have suggested the use of saliva for measurement of markers of oxidative stress Srivastava et al. found, statistically non-significant increasing trend of product of lipidperoxidation in clinic pathological stages of leukoplakia in stages I and II and a significantly decrease in levels of GSH, GPx, catalase, and SOD in patients with leukoplakia compared to healthy control groups.[33]

**Oral cancer**

Oral cancer, well-defined as oral squamous cell carcinoma (OSCC), is one of the most common types of cancer in the world, with delayed clinical detection, poor prognosis, Tobacco, and alcohol are considered the main etiological factors in oral carcinogenesis; moreover, human papillomavirus infection, exposure to radiation and chemicals, and family history of cancer are considered other risk factors for oral cancer. The most common sites for the presentation of oral cancer are the tongue, floor of the mouth and lower lip.
Reactive oxygen species can cause tissue damage by the following mechanisms,

- Lipid peroxidation
- Protein damage
- DNA damage
- Stimulation of pro-inflammatory cytokine
- Oxidation of antiproteases

Several studies have been conducted on the importance and relevance of oxidative stress in OSCC. Rathan Shetty *et al*, did their study on serum antioxidant capacity in oral cancer development but could not find any significant results, Srivastava *et al*. after their study reported that ROS and their deleterious consequences such as lipid peroxidation, have been implicated in the pathogenesis of oral cancer. Metgud et al., reported the diagnostic efficacy of saliva in evaluating levels of MDA and GSH in smoking patients with OSCC. And showed a significant increase of MDA and reduction of GSH progressively from healthy controls to pre-cancerous and topatients with OSCC. For OSCC, as for most other cancer types, the prognosis depends largely on lifestyle factors, medical comorbidity, grading and tumor staging.[34][35]

**CONCLUSION**

In this review, the association between the most frequent inflammatory, potentially malignant and malignant diseases of the oral mucosa and oxidative stress is narrated. Although the molecular mechanisms underlying the etiology of these pathologies still need to be explained and understood in depth, evidence that oxidative stress plays an important role regarding the pathogenesis of oral diseases has been presented.

## References


1. Horwood, G., *et al*. (2023). "It's important to manage our stress": Mental health advice in the Australian print news media during the COVID-19 pandemic. *SSM - Mental Health*, 3, 100204.
2. H&D srl. (Year). *The measurement of oxidative stress* [Description of the document or presentation]. Publisher.
3. Ros, A., et al. (Year). *Oxidative Stress and Disease*. DOI: 10.5772/653663.
4. Khot, V. S., et al. (2018, July 17). *Free Radicals, Oxidative Stress and Diseases: An Overview*.
5. Yang, et al. (2023, May 11). *Effects of an On-Campus Meditation Course on Depression, Anxiety, Stress, and Sleep Quality among South Korean Paramedic Students*.
6. Anhar, et al. (2022). *Stress among Medical Students in the Gaza Strip*. *Journal Title*, Volume (Issue), Page Range.
7. Dmytro, et al. (2012, April 25). *Oxidative Stress: Cause and Consequence of Diseases*.
8. Author(s). (2019). *Role of oxidative stress in biological systems*. *Journal Title*, Volume (Issue), Page Range.
9. Author(s). (2020, July). *Lifestyle, Oxidative Stress, and Antioxidants: Back and Forth in the Pathophysiology of Chronic Diseases*. *Journal Title*, Volume(Issue), Article Number.
10. Forman, H. J. (2021, September). *Targeting oxidative stress in disease: promise and limitations of antioxidant therapy*. *Journal Title*, Volume (Issue), Page Range.
11. Vinay Kumar, et al. (2015). *Role of oxidative stress in various diseases: Relevance of dietary antioxidants*. *The Journal of Phytopharmacology*, 4(2), 126-132.
12. Ahmadi, M., et al. (2011). *Oxidative Stress and Antioxidants*. *DJH*, 3(1).
13. Almokhtar, et al. (2019). *Oxidative stress and antioxidant mechanisms in human body*. *Journal of Applied Biotechnology*, 6(1), 43-47.
14. Ichiishi, E. (2016). *Oxidative Stress and Diseases: Current Topics and Perspective with Clinical Application in Japan*. *J Aging Sci*, Volume (Issue).
15. Nagi, R. (2021). *Therapeutic Role of Phytochemicals in the Prevention of Oral Potentially Malignant Disorders and Oral Cancer – A Review*. *J Evolution Med Dent Sci*, 10(16), 1156-1165.
16. Ghezzi, P. (2017). *The oxidative stress theory of disease: levels of evidence and epistemological aspects*. *British Journal of Pharmacology*, 174, 1784–1796.
17. Liguori, I. (2018). *Oxidative stress, aging, and diseases*. *Clinical Interventions in Aging*, 13, 757–772.
18. Kumar, V., et al. (2015). *Role of oxidative stress in various diseases: Relevance of dietary antioxidants*. *The Journal of Phytopharmacology*, 4(2), 126-132.
19. Bar-Or, D. (2015). *Oxidative stress in severe acute illness*. *Redox Biology*, 4, 340–345.
20. Pizzino, G. (2017). *Oxidative Stress: Harms and Benefits for Human Health*. *Oxidative Medicine and Cellular Longevity*, Volume, Article ID 8416763.
21. Leyane, T. S. (2022). *Oxidative Stress in Ageing and Chronic Degenerative Pathologies: Molecular Mechanisms Involved in Counteracting Oxidative Stress and Chronic Inflammation*. *Int. J. Mol. Sci.*, 23, 7273.
22. Gospodaryov, D. (2012, April 25). *Oxidative Stress: Cause and Consequence of Diseases*.
23. Sarawootpalipoch. (2015). *Oxidative Stress-Associated Pathology: A Review*. *Sains Malaysiana*, 44(10), 1441–1451.

24. Sardaro, N., et al. (2019). *Oxidative Stress and Oral Mucosal Diseases: An Overview*. *in vivo*, 33, 289-296.
25. Aravind Kumar, et al. (Year). *Oxidative Stress and Oral Diseases*. DOI: 10.1007/978-981-15-1568-2_1.
26. Kumar, J., et al. (2017, September). *Oxidative Stress in Oral Diseases: Understanding Its Relation with Other Systemic Diseases*. *Journal Name*, Volume (Issue), Article Number.
27. Miricescu, D. (2011). *Oxidative stress - a possible link between systemic and oral diseases*. *Farmacia*, 59(3).
28. Palla, S. (2015). *Determinants of Malignant Transformation Of Oral Potentially Malignant Disorders – Covering The Gaps*. *Journal of Scientific Dentistry*, 5(1).
29. Aparna, H., et al. (2016, January). *Oxidative Stress in Oral Diseases*. *Oral Dis.*, 22(1), 9–18.
30. Okezie, I. (1998). *Free Radicals, Oxidative Stress, and Antioxidants in Human Health and Disease*. *JAOCS*, 75(2).
31. José Manuel, et al. (2016). *Oxidative stress in the oral illnesses*. *Rev Cubana Estomatol*, 53(4).
32. Verónica. (2022). *Oxidative stress in saliva induced by tobacco smoke: impact on periodontitis and perspectives with redox pharmacology*. *Odontoestomatología*, 24.
33. Bahareh, et al. (2021). *Salivary Oxidative Stress Markers' Relation to Oral Diseases in Children and Adolescents*. *Antioxidants*, 10(10), 1540.
34. L'ubomíra Tóthová, et al. (2015, October). *Salivary markers of oxidative stress in oral diseases*. *Frontiers in Cellular and Infection Microbiology*, 5, Article 73.



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