IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

# OXIDATIVE STRESS: A REVIEW

Surbhi Chourasiya[1], Preeti Tirkey[2], Malabika Sikdar[3]
Ph.D. Research Scholar[1,2], Associate Professor[3]
Department of Zoology
Dr. Harisingh Gour Vishwavidyalaya (A Central University), Sagar (M.P.)

## Abstract

The term "oxidative stress" refers to an imbalance in the body's antioxidant enzymes between free radicals and their stabilizing agents. Normal cellular metabolism can generate reactive oxygen species, also known as free radicals, which interact with biomolecules including protein, lipid, and DNA to harm cells and bring about deteriorative changes. Free radicals are essential for cellular communication and physiological control at low concentrations, but at excessive concentrations, they can harm cells. In contrast, antioxidants reduce oxidants by giving their own electron to stabilize free radicals and turn them into non-reactive compounds in order to reduce the negative effects that these radicals have on the cell. Chronic disorders including cancer, diabetes, neurodegenerative diseases, and cardiovascular diseases all have oxidative stress as a major contributor to their etiology. Increasing amounts of pro-oxidant substances over time might result in structural flaws at gene expression anomalies brought on by functional changes at the mitochondrial DNA level, as well as in a number of enzymes and cellular structures. The current way of life, which includes eating processed food, being exposed to a variety of chemicals, and not exercising, is a significant factor in the development of oxidative stress. However, the capacity of medicinal plants with antioxidant capabilities to cure or prevent a number of human illnesses in which oxidative stress appears to be a contributing factor has been utilized. The diseases that oxidative stress is one of the triggers for are discussed in this overview, along with the antioxidant chemicals obtained from plants and their processes of antioxidant defenses that can aid in the prevention of these diseases. The impacts of antioxidant compounds employed to lower oxidative stress in a number of pathological dysfunctions in both positive and negative conditions are finally reviewed.

**Keywords:** Oxidative Stress, Antioxidant, Free Radicals, ROS, Chronic disorders.

## I. Introduction

Aging is the process through which a person's body gradually alters. Reactive oxygen species (ROS), which induce lipid breakdown, DNA damage, collagen breakdown, protein unraveling, and fragmentation are to blame for these alterations (Said, 2014). Ultimately, this will result in cell death. We age and appear older as a result of apoptosis and aging. At the tissue and cellular levels, free radicals (produced from oxygen) are likely to be responsible for the damaging consequences of aging**.** Given that free radical reactions are known to accelerate aging, it is recommended that efforts be taken to limit them, which should be able to slow the generation of aging changes and result in an ongoing decline in the pace of aging and age-related alterations (Fusco et al., 2007). Free radicals are unpaired electrons that are very reactive by nature and injure our bodies. According to (Lobo et al., 2010), the primary causes of ROS formation include phagocyte cells, damaging UV radiation, metabolism, and environmental pollutants. These sources increase oxidative stress in the body by producing free oxygen radicals. According to the notion of oxidative stress, the stress results in cellular

deterioration. It ultimately results in cell death by starting a series of cell death processes. Oxidative damage caused by free radicals also leads to the generation of superoxide radicals, peroxynitrite, and many other radicals, and is a key contributor to how we age and the emergence of age-related illnesses including Alzheimer's, Parkinson's, neurodegenerative diseases, and other ailments. For a living thing to survive, oxygen is absolutely necessary. It also serves as the foundation for cellular respiration, which results in aerobic respiration. But oxygen operates as a chemical or molecule that generates oxidative stress in the form of a free radical (Stamati et al., 2011). a variable stage of Oxidative stress is the production and removal of free radicals, which causes stress. Free radicals produced in high quantities inside the human body under these circumstances led to oxidative stress, which has a negative impact on the life cycle of living cells and can lead to illness (Bhattacharya et al., 2015). Consequently, these oxidative stressors contribute to a number of serious age-related illnesses, including cancer, neurodegeneration, and aging. Oxidative stress refers to the negative consequences of ROS that harm living things. Oxidative stress occurs when the body's number of antioxidants declines and ROS generation increases. Ozone, singlet oxygen, superoxide, hydroxyl radicals, hydrogen peroxide, hydroperoxyl radicals, and others are examples of reactive oxygen species (Halliwell and Cross, 1994). A cell experiences oxidative cellular stress if the balance between oxidative stress producers such as free radicals and antioxidants is disturbed.

## II. Oxidative Stress

Free radicals represent a major threat to homeostasis and, as a result, to the health of aerobic organisms since they are chemical substances with one or more unpaired electrons in their outer orbitals (Wong et al., 2012). Free radicals made of oxygen can arise during a variety of routine metabolic activities. Free radicals (FR), despite the fact that they have the potential to damage the organism, are created naturally throughout a number of metabolic processes. The main endogenous free radical generators include oxidant enzyme activities, the mitochondrial electron transport chain, the microsomal membrane electron transport chain, and auto-oxidation mechanisms (Freeman et al., 1982; Hauck et al., 2000; Matés et al., 2002). In any condition when there is a large imbalance between the creation of FR or reactive oxygen species (ROS), also known as the oxidative load, and the antioxidant defense system, it is referred to as being under oxidative stress. Sies came up with the term in 1985. The oxidative load is to blame. The steady-state concentration of reactive oxygen or oxygen radicals in a biological system is determined (Baynes, 1991). According to a definition given by the term "oxidative stress," it is "a disruption in the prooxidant-antioxidant equilibrium favoring the former, resulting to potential damage"(Sies, 1985). By up-regulating their reductive defense mechanisms and resetting the oxidant/antioxidant balance, cells can endure mild oxidative stresses. But when enzyme defects or substrate shortages prevent this enhanced synthesis from occurring, Oxidative stress develops when an imbalance continues due to an excessive oxidative burden (Halliwell, 2015). The highest level of bioactivity is seen in reactive oxygen species (ROS), which also comprise non-radical oxygen species like hydrogen peroxide ($H_2O_2$) and superoxide and hydroxyl radicals. The production of ROS is particularly high in activated neutrophils, monocytes, smooth muscle cells, and endothelial cells, although all cells are capable of it, depending on the degree of aerobic metabolism (Nanda et al., 2007). Inactivation causes oxidative stress, which occurs between the production of ROS. ROS also damages the fundamental cell structures. Large molecules, such as lipid, protein, and DNA molecules, with which they readily interact, tear down cell membranes and activate or inactivate enzymes (Niki et al., 1995). These defenses, which include endogenous and exogenous antioxidants produced by the body, are both enzymatic and non-enzymatic (Hayes and McLellan, 1999; Sies and Medicine, 1999). Either neutralize or detoxify ROS, stop their production, sequester the transition metals that produce free radicals, or neutralize or detoxify ROS. Exogenous sources are ones that are obtained through food (Benzie, 1999; Yao et al., 2004). By transforming free radicals into less damaging molecules, antioxidant enzymes work to neutralize them (Stahl and Sies, 2003). Catalase (CAT), glutathione reductase (GR), glutathione peroxidase (GPx), and superoxide dismutase (SOD). If you want to know more about this, go here. SOD catalysis the superoxide anion radical dismutation reaction, which yields peroxide ($H_2O_2$) and molecular oxygen (O2). The hemoprotein catalase, which includes iron, converts hydrogen peroxide into water and oxygen (Kurasaki et al., 1986). Reduced glutathione (GSH), required by

the enzyme GPx for the catalytic activity, is provided by glutathione reductase. A cofactor for the enzyme GPx is the selenium ion (Mézes and Balogh, 2009). GPx is one of the most potent antioxidants found in erythrocytes. As $H_2O_2$ levels rise as a result of decreased GPx function, there is severe cellular damage that results (Rybka et al., 2013). One of the most important roles of non-enzymatic antioxidants such glutathione, tocopherols, retinols, and ascorbate is scavenging ROS (Kefer et al., 2009).

## III. Intoxication of Oxygen Species

In metabolic processes, oxygen is utilized. When it interacts with the metabolic molecule, it occasionally creates a free radical that is harmful to living beings (Rahman et al., 2012). Although one electron can be obtained or lost in an oxygen molecule or one of its constituents, oxygen can nevertheless change from a stable material into a reactive oxygen species. The harmful consequences of oxygen synthesis are referred to as "oxygen toxins." The imbalance caused by reactive oxygen species (ROS) converting from one form to another while devouring a cell's or a biomolecule's beneficial component is referred to as "oxidative stress" (Kuttappan et al., 2021). Reactive oxygen species (ROS) are key players in a number of biological activities, including the oxidative burst response required for phagocytes. ROS are also involved in a number of cells signaling pathways. Free radicals are oxygen-containing molecules with an uneven number of electrons. They have an uneven amount, which makes them easily interact with other molecules. Because they interact with other molecules so fast, free radicals can result in the formation of long chemical chains in your body. These actions are known as oxidations. Pro-oxidant chemicals, which either produce reactive oxygen species or impede antioxidant processes, are responsible for oxidative stress (Puglia and Powell, 1984). These substances can cause oxidative stress, which can harm cells and tissues. For instance, an overdose of the painkiller paracetamol (acetaminophen) can cause catastrophic liver damage, in part because it produces reactive oxygen species. (Hinson et al., 1981; Lemasters, 2002) Various Reactive Oxygen Species are shown below in the Table. 1.

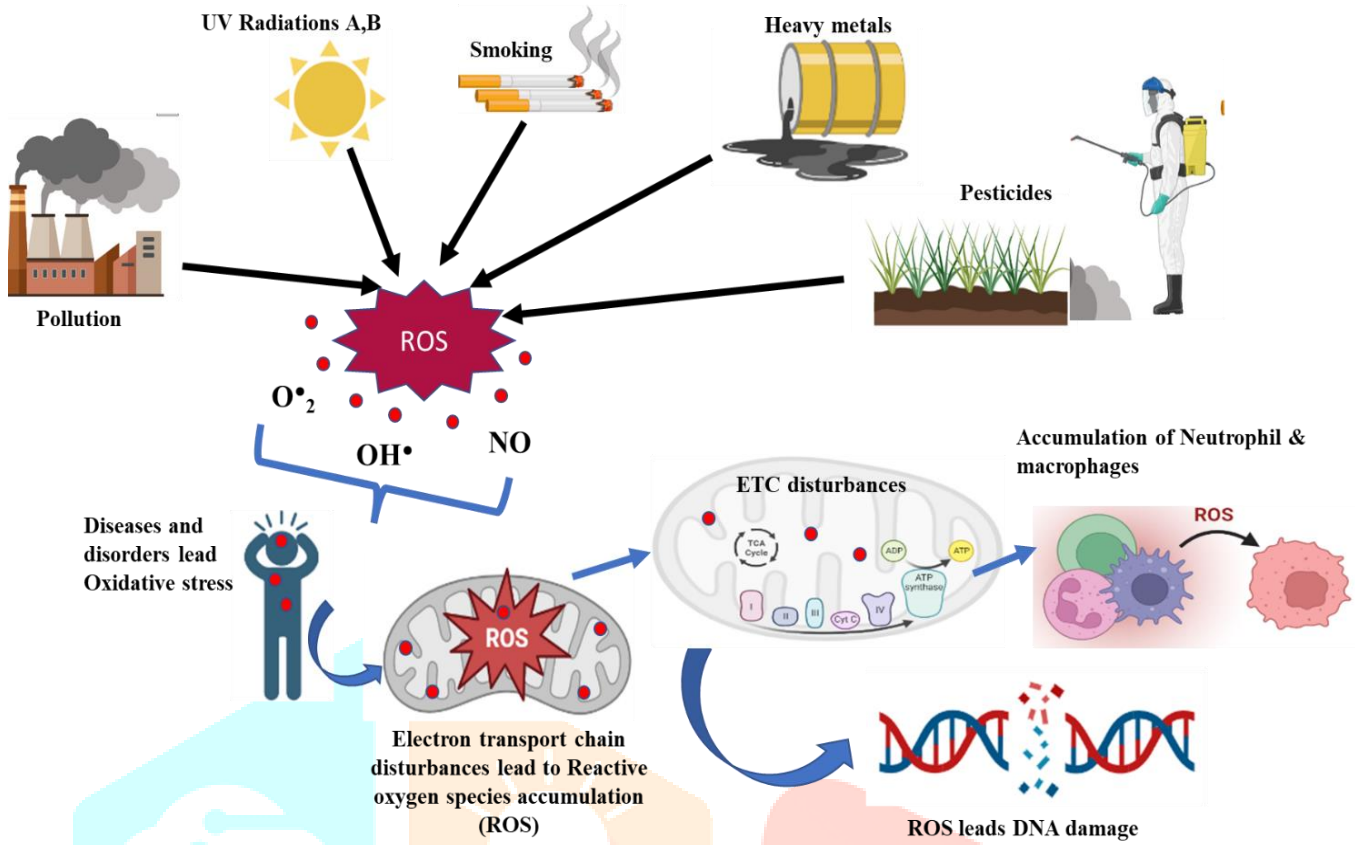
**Table 1. Showing the different Oxidants and Pro-oxidants**

| Oxidants (Free Radicals) | Pro-oxidants |
|---|---|
| $OH^{\cdot}$ (Hydroxyl radical) | $O_2$ (Singlet oxygen) |
| $O_2^{\cdot}$ (Superoxide radical) | $H_2O_2$ (Hydrogen peroxide) |
| $RS^{\cdot}$ (Thyl radical) | HOCl (Hypochlorous acid) |
| $RO_2^{\cdot}$ (Peroxyl radical) | LOOH (Lipid peroxide) |
| $RO^{\cdot}$ (Alkoxyl radical) | $O_3$ (Ozone) |
| $LOO^{\cdot}$ (Lipid peroxyl) | |

## IV. ROS-Linked Illnesses and Abnormalities

Reactive oxygen species are an immediate byproduct of normal cellular metabolism and play a crucial role in signaling networks inside cells. However, under some circumstances, an excessive buildup of ROS can lead to a pathological situation that is the cause of a number of illnesses (Fanjul-Moles et al., 2016). Reactive oxygen species created by oxidative stress include superoxide, hydroxyl, and hydrogen peroxide. Since most oxidative DNA damage is indirect, radical adduction to DNA can cause mutation and the growth of cancerous cells. Mitochondrial ETC is one type of internal source factor for the generation of free radicals in internal cellular metabolism (Shinde et al., 2012). Some of these reactive oxygen intermediates may also serve as secondary redox signaling mediators. This might disrupt typical cellular signaling networks. Certain herbicides, such rotenone, increased the production of ROS and inhibited complex 1 of the mitochondrial electron transport chain (ETC), increasing the risk of brain-related illnesses in humans (Swarnkar et al., 2010). Additionally, a number of other environmental toxins, such as UV radiation, ionizing radiation, pesticides, pollution, heavy metals, and smoke from cigarettes, among others, have a negative impact on people's daily lives. All over generation of radicals an oxidative abnormalities mechanistic pathway shown in the figure 1.

**Figure 1. Showing Oxidative Stress mechanistic pathway**




## V. Conclusion

Animal cells all experience mortality; not one cell in the animal kingdom is everlasting. A cell can become mortal during the ageing process, which is a normal occurrence. Oxidative stress results in the production of reactive oxygen species, which can lead to a number of pathogenic situations inside of cells. Reactive oxygen species is the molecule in oxygen that causes oxidative stress in the body or cell. Once a sufficient amount of ROS has accumulated in the cell, degenerative conditions develop that result in cellular damage and disease states. The cellular condition known as oxidative stress can, to a certain extent, be stabilized by enzymes that act as antioxidants. Many antioxidants, including glutathione peroxidase, catalase, vitamin E, vitamin C, and metabolic antioxidants like bilirubin and uric acid, help the body's own self-healing mechanisms lower the number of free radicals in the tissue cell. In this review, we investigated that the harmful effects of nutrients and antioxidants on living things for degenerative mitochondrial changes and deteriorative reactions brought on by excessive free radical generation and oxidative stress.

## Acknowledgment:

Author gratefully acknowledge the HOD, Department of Zoology, Dr. Harisingh Gour Vishwavidyalaya, Sagar, Madhya Pradesh for providing infrastructure and experimental facility and also for Non-NET UGC fellowship.

## Conflicts of Interest:

There is no conflict of interest.

## References:


Baynes J. W.." Role of oxidative stress in development of complications in diabetes" J Diabetes ,1991 40, 405-412.

Benzie I. F. "Vitamin C: prospective functional markers for defining optimal nutritional status" J Proceedings of the Nutrition Society, 1999, 58, 469-476.

Bhattacharya, A., Hegazy, A. N., Deigendesch, N., Kosack, L., Cupovic, J., Kandasamy, R. K., Hildebrandt, A., Merkler, D., Kühl, A. A., Vilagos B., "Superoxide dismutase 1 protects hepatocytes from type I interferon-driven oxidative damage" J Immunity, 2015. 43, 974-986.

Fanjul-Moles, M. L., López-Riquelme, Longevity, C. G. O. "Relationship between oxidative stress, circadian rhythms, and AMD" J Oxidative Medicine Cellular Longevity 2016.

Freeman, B. A., Crapo, methods, "Biology of disease: free radicals and tissue injury" J Laboratory investigation a journal of technical methods pathology, 1982 47, 412-426.

Fusco, D., Colloca, G., Monaco, M. R. L., Cesari, M. "Effects of antioxidant supplementation on the aging process" J Clinical interventions in aging, 2007, 2, 377.

Halliwell, B., "Free Radicals and Other Reactive Species in Disease". John Wiley & Sons: Chichester. 2015

Halliwell, B., Cross, "Oxygen-derived species: their relation to human disease and environmental stress" J Environmental health perspectives, 1994.102, 5-12.

Hauck, S. J., Bartke A. "Effects of growth hormone on hypothalamic catalase and Cu/Zn superoxide dismutase" Free Radical Biology Medicine, 2000, 1 (28), 970-978.

Hayes, J. D., McLellan, "Glutathione and glutathione-dependent enzymes represent a co-ordinately regulated defence against oxidative stress" J Free radical research, 1999, 31, 273-300.

Hinson, J. A., Pohl, L. R., Monks, T. J., Gillette J. R., "Acetaminophen-induced hepatotoxicity" J Life sciences, 1981. 29, 107-116.

Kefer, J. C., Agarwal, A., Sabanegh E., " Role of antioxidants in the treatment of male infertility" J International journal of Urology, 2009, 16, 449-457.

Kurasaki, M., Saito, T., Kaji, H., Kojima, Y., Saito K., "Increased erythrocyte catalase activity in patients with hyperthyroidism" J Hormone metabolic research, 1986, 18, 56-59.

Kuttappan, V. A., Manangi, M., Bekker, M., Chen, J., Vazquez-Anon M. J., "Nutritional intervention strategies using dietary antioxidants and organic trace minerals to reduce the incidence of wooden breast and other carcass quality defects in broiler birds" J Frontiers in Physiology, 2021, 12, 663409.

Lemasters, "Mechanisms of hepatotoxicity" J Toxicol Sci, 2002, 65, 166176

Kobayashi. Lobo, V., Patil, A., Phatak, A., Chandra N., "Free radicals, antioxidants and functional foods: Impact on human health" J Pharmacognosy reviews, 2010, 4, 118.

Matés, J. M., Pérez-Gómez, C., de Castro, I. N., Asenjo, M., Márquez J., "Glutamine and its relationship with intracellular redox status, oxidative stress and cell proliferation/death" J The international journal of biochemistry cell biology, 2002, 34, 439-458.

Mézes, M., Balogh, K. "Prooxidant mechanisms of selenium toxicity–a review" J Acta Biologica Szegediensis, 2009 53, 15-18.

Nanda, N., Bobby, Z., Hamide, A., Koner, B. C., Sridhar, "Association between oxidative stress and coronary lipid risk factors in hypothyroid women is independent of body mass index" J Metabolism 56, 1350-1355.

Niki, E., Noguchi, N., Tsuchihashi, H., Gotoh N., "Interaction among vitamin C, vitamin E, and beta-carotene" J The American journal of clinical nutrition, 1995, 62, 1322S-1326S.

Puglia, C. D., Powell, S. R. "Inhibition of cellular antioxidants: a possible mechanism of toxic cell injury" J Environmental health perspectives, 1984, 57, 307-311.

Rahman, T., Hosen, I., Islam, M. T., Shekhar, Oxidative stress and human health. H. U., 2012.

Rybka, J., Kędziora-Kornatowska, K., Banaś-Leżańska, P., Majsterek, I., Carvalho, L. A., Cattaneo, A., Anacker, C., Kędziora, "Interplay between the pro-oxidant and antioxidant systems and

proinflammatory cytokine levels, in relation to iron metabolism and the erythron in depression" J Review Article: Oxidative stress versus., Medicine, 2013, 63, 187-194.

Said, M. "Al-Dalaen, Aiman I. Al-Qtaitat" J Review Article: Oxidative stress versus., 2014.

Shinde, A., Ganu, J., Naik, P. J. "Effect of free radicals & antioxidants on oxidative stress: a review" J Journal of Dental, 2012. 1, 63.

Sies, H., "Oxidative stress: introductory remarks. [In] Sies H.(ed.) Oxidative Stress". Academic Press: London. 1985.

Sies, H. " Glutathione and its role in cellular functions" J Free Radical Biology, Medicine, 1999, 27, 916-921.

Stahl, W., Sies, H. "Antioxidant activity of carotenoids" J Molecular aspects of medicine, 2003, 24, 345-351.

Stamati, K., Mudera, V., Cheema, "Evolution of oxygen utilization in multicellular organisms and implications for cell signalling in tissue engineering" J Journal of tissue engineering, 2011, 2, 2041731-411432365.

Swarnkar, S., Singh, S., Mathur, R., Patro, I., Nath, C., " A study to correlate rotenone induced biochemical changes and cerebral damage in brain areas with neuromuscular coordination in rats" J. Toxicol., 2010, 272, 17-22.

Wong, D. M., Moore, R. M., Brockus, C. W. "Mechanisms of oxidative injury in equine disease" J Compend Contin Educ Vet, 2012. 34, E6.

Yao, L. H., Jiang, Y.-M., Shi, J., Tomas-Barberan, F., Datta, N., Singanusong, R., Chen, S. "Flavonoids in food and their health benefits" J Plant foods for human nutrition, 2004, 59, 113-122.