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Review Article

The Role of Oxidative Stress and Reactive Oxygen Species in Human Disease: Mechanisms, Consequences, and Preventive Strategies

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Abstract

The imbalance between the reactive oxygen species and the antioxidant defenses of the body defines a condition known as oxidative stress, which causes cellular damage and is involved in the pathogenesis of diseases such as cancer, cardiovascular diseases, and neurodegenerative disorders. ROS are generated in normal metabolic conditions, especially in mitochondria, and an overproduction of ROS causes damage to DNA, proteins, and lipids, accelerating tissue dysfunction and aging. On the other hand, exogenous factors include those associated with the environment, inflammation, and lifestyle that tip the balance towards ROS production. Representative enzymatic antioxidants are superoxide dismutase, catalase, and glutathione peroxidase; representative non-enzymatic antioxidants include vitamins C and E. Such all aid in neutralizing ROS and are thus protective of health. It is thus suggested that OS is implicated in the pathogenesis of Alzheimer's, Parkinson's, and cardiovascular disorders, whereby it promotes inflammation, DNA damage, and functional loss in cellular homeostasis. Prevention of oxidative stress involves the enhancement of antioxidant mechanisms, minimization of environmental pollution, and a healthy lifestyle, including adequate exercise and proper nutrition. There are also some approaches on how pharmacological agents can activate mechanisms supporting the body's defense against oxidative damage, such as gene regulation strategies like the activation of the Nrf2 pathway. The key to minimizing diseases associated with oxidative stress, for better health and longevity, may lie in maintaining a critical balance between the production of ROS and antioxidant activity.

Keywords: *Oxidative Stress; Antioxidants; Neurodegenerative Diseases; Cancer; Cardiovascular Diseases (CVD); Inflammation*

Introduction

The generation of reactive oxygen species (ROS) overwhelms the body's antioxidant defenses creating oxidative stress which results in cellular damage and promotes disease development. Highly reactive molecules with unpaired electrons known as ROS emerge naturally during standard metabolic activities, particularly within mitochondria during cellular respiration. A variety of environmental elements including pollution, ultraviolet radiation, cigarette smoke, and specific dietary components intensify ROS production which overwhelms the body's antioxidant defenses leading to oxidative stress. DNA, proteins, and lipids sustain damage from this imbalance which then disrupts cellular function while speeding up the aging process. The emergence of oxidative stress as a critical factor drives the development of numerous major diseases such as cardiovascular disorders, neurodegenerative diseases, cancer, and autoimmune conditions. The body's

ability to sustain cellular integrity and halt disease advancement relies on its capacity to boost antioxidant mechanisms while controlling ROS generation.

The role of oxidative stress and free radicals in human diseases

This short overview introduces the reader to what oxidative stress is, some of its aspects, and its involvement in pathology, including a brief overview of processes and consequences of oxidative stress in whole cells, tissues, organs, and organisms.

Oxidative stress can be described by the attack of free radicals on cells. This process involves species oxygen (ROS) and nitrogen (RNS) reagents, so-called free radicals; their toxicity; and the imbalance of production of reactive oxygen species and reactive nitrogen species and antioxidant defense system [1,2].

Oxidative stress at the cellular level is a very different kind of concept from psychological stress.

Oxidative stress is implicated in DNA, proteins, and lipids alterations and their consequences in nervous system disorders, heart, lungs, kidneys, liver, etc., and also could be related to diseases involving more than one organ and systemic diseases as well as cancer and diabetes [3,4].

Pathogenesis studies with accuracy point out that a wide range of diseases is associated with oxidative stress and reactive oxygen species [3,4]. Many diseases resulting from inflammation could actually be caused by oxidative stress itself [5-7]. Diseases originating either in the brain [8-10] or those with no apparent pathogenic etiology have also been associated with oxidative stress [11-13].

New methods are being developed to detect the formation of ROS in cells and blood, not only during disease processes but even before symptoms appear [14-17].

Excessive amounts of ROS are either produced in the body due to host defense mechanisms against infection or produced at normal levels but not neutralized because of the insufficient antioxidant capacity of the homeostasis system [18]. Let us remember that, in biology as well as in systems science, homeostasis is the feature of a phenomenon in which a key factor. Herein, it is the redox status or the amount of ROS is maintained around a value which is valuable for the considered system, thanks to a regulation process.

ROS are generated as intermediates and by-products in the cycle of energy production in mitochondria, the respiratory chain, and the endoplasmic reticulum [19]. The ROS generation also occurs in neutrophils and macrophages during inflammation and other normal cellular metabolic activities [18].

Free radicals are produced in response to certain conditions of the environment: after exposure to ultraviolet radiation, intake of tobacco, alcohol or narcotics, long contact with environmental pollutants-strong oxidizers, heavy metal ions, herbicides and pesticides, photosensitizers, etc., UV light or ionizing radiation and usual cellular production in physiological processes [13].

These include enzymes like Superoxide Dismutase-SOD [20], glutathione peroxidase-GPX, catalase-CAT, and many other enzymatic and non-enzymatic systems that protect the cells against the harmful effects of ROS. To stop the propagation of mutations, several mechanisms of DNA repair and initiation of apoptosis-cell death-have been developed at the cellular level [21].

Brief episodes of oxidative stress-what might be termed “oxidative shock”-can themselves have therapeutic effects on the aging phenotype via the induction of endogenous antioxidative defenses. Thus, for example, ozone therapies have shown some benefit to redox status in elderly patients with cardiovascular disease to ameliorate chronic oxidative stress [22].

However, such cases are not out of controversy since even a low dose of ozone can cause cancer. In the same way, increasing the levels of ROS after moderate sports activity in older people with chronic oxidative stress is generally effective in improving cellular antioxidant responses [23].

Another contentious issue is the action of antibiotics in killing bacteria by an oxidizing mechanism. Though some believe that antibiotics kill bacteria through induction of ROS, other researchers have shown that no generation of ROS occurs upon the attack of pathogens and thus lethality is likely a consequence of inhibition of protein synthesis, DNA replication, and other vital processes [24-27].

In summary, the production of ROS must be subject to sustainable control so that a redox homeostasis is guaranteed. Redox homeostasis is an active process that provides a counterbalancing effect by antioxidants so as to avoid sliding into oxidative stress or immune deficiency.

Maintaining it depends on a healthy body. All living organisms have such homeostasis and the mechanisms for maintaining it [18].

The regulation of ROS production is complex, with many pathways involved: i) autonomous replenishment of antioxidants stored and synthesized in different tissues and cells; ii) many sulfur-containing proteins that can act as free radical scavengers-it should be noted that these proteins can easily be modified by the biological environment, a very important fact for the control of protein functions-and finally iii) some small bimolecular antioxidants such as glutathione, vitamin E, and vitamin C, which act as buffers for free radicals interacting with various cellular components and influencing defense organization, enzymatic activity, as well as cell growth and development by modulating mitosis, senescence, and cell death. These antioxidants may also impact the expression of genes that are induced by stress responses in optimizing cellular defenses [28].

Notably, cellular energy production results in the generation of a great amount of free radicals, some of which can cause damage to nucleic acids, proteins, and lipids. Thus, in concert with exogenously derived oxidants, internally generated ROS can generate oxidative stress that the human body must cope with to survive [28].

Prolonged oxidative stress may result in severe disease and accelerated aging. Many powerful exogenous oxidants, produced or used in various industrial processes, are then released into the environment, creating very serious pollution problems. Cited are chromium, chlorine, ozone, nitrogen oxides and others [29-32]. For example, highly oxidizing chromate and bichromate species, now banned in Europe and the United States, are mutagenic, carcinogenic, and cause DNA damage. Along with perchlorates and common compounds like hypochlorites and chlorine commonly used as bleaching agents and for water disinfection. Some pesticides and herbicides are also powerful oxidants [33,34] in paraquat for example, previously used under license in some countries like the United States, created a serious health threat depending of type of the exposure. Acute symptoms may initiate with pulmonary insufficiency and then pulmonary fibrosis. Very low dose and in presence of hydrogen peroxide, the paraquat has been shown to cause DNA damage [30].

Reactive oxygen species are formed as by-products of various reactions of exogenous oxidants mainly inhaled oxygen from air and fuels food and nutrients. In normal conditions, the generation of ROS remains well balanced by homeostasis mechanisms and well controlled by enzyme-catalyzed reactions. Total reduction of molecular oxygen requires transmission four electrons. This process is catalyzed by enzymes step by step, producing a variety of intermediates, including one free radical-the superoxide anion-neutralized by antioxidants such as glutathione (GSH) and Superoxide Dismutase. SOD deficiency causes various mutations and various disorders such as neurodegeneration, lactic acidosis, myopathy, muscle atrophy, cataracts, reduced life span, decreased fertility, liver cancer etc [28].

Some free radicals, such as HO•, are very noxious for biological materials, in which they can cause damage to nucleic acids, proteins, and lipids. Thus, at the DNA level, strand breaks, base oxidation, among others, might be induced. These lesions may not be repairable and could eventually lead to mutations and carcinogenesis. Generation of HO• radicals is a host defense function against pathogens; such generation, if not promptly neutralized, may also be harmful to the host. Fortunately, HO• radicals have an extremely short life-a few nanoseconds-to the extent that they cannot diffuse far from their site of formation [28].

Cardiovascular diseases (CVD)

Oxidative stress is considered a major forcing factor in the development and progression of CVD. ROS, especially superoxide anions (O_2^-), have a central role in endothelial dysfunction, which characterizes atherosclerosis. The endothelial cells, lining the wall of blood vessels, are responsible for the regulation of vascular tone and promoting blood flow. ROS impede the function of endothelial cells by reducing the bioavailability of NO, a key molecule in the induction of vasodilation, as stated by Valko., *et al.* (2007). Furthermore, oxidative stress leads to the occurrence of lipid peroxidation and the generation of ox-LDL, thereby eliciting an inflammatory response and resulting in plaque build-up within arterial walls. This process lies at the heart of atherosclerosis pathogenesis, leading to potential endpoints like myocardial infarction and stroke [1].

The role of oxidative stress in hypertension is also one of the most highly researched. High levels of ROS induce contraction in the vascular smooth muscle cells and enhance vasoconstriction, adding to peripheral vascular resistance representative of hypertension [1]. It also enhances inflammatory cascade in hypertension, further leading to impairment of vascular function.

Neurodegenerative diseases

In neurodegenerative diseases, including Alzheimer's disease (AD) and Parkinson's disease (PD), oxidative stress plays a critical role in the pathophysiology of neuronal degeneration. In AD, ROS-induced oxidative damage contributes to the aggregation of amyloid-beta (Aβ) peptides, leading to the formation of amyloid plaques, a pathological hallmark of the disease (Markesbery, 2005). Oxidative stress is also implicated in the hyperphosphorylation of tau protein, leading to the formation of neurofibrillary tangles that disrupt neuronal function and cause cognitive impairment. Besides, oxidative damage to mitochondrial DNA, proteins, and lipids in neurons exacerbates cellular dysfunction, leading to neuronal death (Swerdlow., *et al*. 2014).

Similarly, in PD, the oxidative stress is considered to take part in the selective degeneration of dopaminergic neurons in the substantia nigra, a part of the brain which is very important for motor control. The accumulation of ROS in these neurons leads to lipid peroxidation, mitochondrial dysfunction, and apoptosis, contributing to the motor deficits characteristic of the disease (Jenner, 2003). Notably, the mitochondrial dysfunction in PD is considered a key player in the excessive production of ROS, thus creating a vicious circle leading to oxidative damage and death of neurons (Müller., *et al*. 2004).

Cancer

Cancer is one of the diseases where oxidative stress has an important role. The ability of ROS to induce DNA damage may result in mutations, genetic instability, and promote tumor initiation and progression. Free radicals can cause base modifications, strand breaks, and chromosomal instability that may lead to mutations in oncogenes or tumor suppressor genes (Bergamini., *et al*. 2004). Such genetic damage has particular relevance to the initiation phase of tumor development. Besides, chronic oxidative stress supports a pro-inflammatory environment that favors tumor growth and metastasis. For instance, ROS may trigger a number of signaling pathways, including nuclear factor kappa B (NF-κB), which further contribute to inflammation and tumor progression (Sahin., *et al*. 2014).

ROS also contribute to angiogenesis, a process through which tumors stimulate the growth of new blood vessels in support of their rapid growth. Activation of hypoxia-inducible factors, induced by ROS, may trigger the expression of pro-angiogenic factors like vascular endothelial growth factor (VEGF) and promote vascularization of tumors (Sies 2015).

Autoimmune diseases

There is a close link in the involvement of oxidative stress in autoimmune disease pathogenesis. Diseases that might be under the shadow of the same principle of oxidative stress as an underlying etiology are RA and SLE. Oxidative stress activates the synovial fibroblast and contributes to inflammation by overproduction of TNF-α and degradative changes of the cartilage in RA [10]. Besides that, the oxidative modifications of proteins, lipids, and nucleic acids in joints mediated through ROS come out with an autoimmune response-a self-attack of the immune system on the tissues.

In SLE, disease pathogenesis is also significantly associated with oxidative stress. ROS can activate both autoreactive T cells and B cells, leading to the production of autoantibodies that target the body's own tissues, especially in the kidneys and skin (Yoshida., *et al*. 2014). The resulting inflammation and tissue damage are big contributors to the clinical features of SLE, such as renal failure and cutaneous lesions.

Other conditions: Acute and chronic inflammation

Oxidative stress has been implicated in various acute and chronic inflammatory conditions, including ischemia-reperfusion injury, sepsis, and chronic obstructive pulmonary disease. Ischemia-reperfusion injury represents the restoration of blood flow to previously ischemic tissues, resulting in a burst of ROS production. These ROS cause oxidative damage to cellular membranes, proteins, and DNA, thus exacerbating tissue injury and inflammation (Pacher., *et al*. 2005). Moreover, in sepsis, ROS overproduction by activated neutrophils contributes to systemic inflammation and then to organ failure (Cohen, 2002).

In COPD, oxidative stress is thought to result from both environmental factors (such as cigarette smoke) and inflammatory cell activation, leading to damage of the lung epithelium, chronic inflammation, and the destruction of alveolar tissue (Rahman., *et al*. 2006). This process contributes to the characteristic airflow limitation and emphysema seen in COPD patients.

Conclusively, oxidative stress and free radicals are implicated in the pathogenesis of a wide range of human diseases. These reactive species result in the destruction of cellular components, genetic mutations, inflammation, and functional impairments in the affected tissues. Since the central role of oxidative stress is considered crucial for disease development, therapeutic strategies that target either the production of ROS or enhancement of antioxidant defense mechanisms do indeed hold immense promise. Such approaches may offer novel interventions in the prevention or treatment of diseases associated with oxidative damage.

Prevention

Oxidative stress is an imbalance between ROS and antioxidant defenses, consequently leading to cellular damage and causing various diseases such as cancer, cardiovascular conditions, and neurodegenerative disorders. According to Halliwell and Gutteridge, 2015, the prevention of oxidative stress is crucial for maintaining cellular health and function. Key pathways and strategies in combating oxidative stress in humans are available both at an endogenous and exogenous level.

Endogenous antioxidant mechanisms

The human body's first line of defense against oxidative stress includes endogenous enzymatic antioxidants such as Superoxide Dismutase (SOD), Catalase (CAT), and Glutathione Peroxidase (GPx). These enzymes work in a sequence to neutralize ROS by converting superoxide radicals into hydrogen peroxide, a less harmful molecule, which, in turn, undergoes further degradation by CAT and GPx enzymes (Sies and Jones, 2020). Glutathione is another critical endogenous antioxidant that plays a crucial role in detoxifying ROS and maintaining cellular redox homeostasis (Jones, 2006).

Dietary antioxidants

Dietary antioxidants also form an important defense mechanism against oxidative stress. Vitamins C and E, carotenoids, flavonoids, and polyphenols are among the major players in neutralizing ROS. Vitamin C scavenges free radicals and regenerates other antioxidants, including vitamin E, which protects cell membranes from lipid peroxidation (Navarro and Boveris, 2007). Carotenoids such as β-carotene and lycopene and polyphenols from berries and green tea are potent antioxidants that help reduce oxidative damages (Ramachandran and Ramasamy 2017).

Gene expression and redox regulation

Besides that, gene regulation is another important mechanism of prevention against oxidative stress. The transcription factor Nrf2 activates AREs to induce the expression of some protective enzymes, such as SOD and GPx, important in maintaining the cell's reducing environment (Mates and Sánchez-Jiménez, 2000). The Nrf2 response to oxidative stress involves its release from the inhibitor Keap1, translocation to the nucleus, and promotion of enzymes synthesis concerned with antioxidant defense (Packer and Cadenas, 2005).

Physical exercise

Moderate physical activity enhances the production of ROS, but at the same time, it increases the body's antioxidant capacity. Physical exercise increases the activity of endogenous antioxidants, like SOD and CAT, by inducing adaptive responses against oxidative stress (Banjanin and Kresović, 2014). However, excessive exercise may overwhelm the body's antioxidant defenses, leading to oxidative damage (Mates and Sánchez-Jiménez, 2000). Therefore, a balanced approach to physical activity is necessary for enhancing antioxidant activity.

Pharmacological interventions

Agents tried to reduce oxidative stress include pharmacological agents such as Nrf2 activators, resveratrol, curcumin, and coenzyme Q10. These are antioxidant compounds that may help in reducing oxidative damage in tissues (Jones, 2006; Halliwell and Gutteridge, 2015). However, further studies are needed to understand the long-term efficacy and safety of these interventions.

Environmental and lifestyle factors

Whereas ROS is generated in small amounts through cellular metabolism, exposure to environmental pollutants like tobacco smoke, UV radiation, and air pollution can substantially increase its production. Decreasing exposure to these elements, coupled with lifestyle decisions involving a diet packed with antioxidants, adequate sleep, and stress management, is crucial for the prevention of oxidative stress. According to Ramachandran and Ramasamy (2017), one should avoid smoking and excessive sun exposure as preventive measures.

To avoid oxidative stress, there must be a fine combination of antioxidant systems, nutrition interventions, modulation of gene expressions, exercise, and lifestyle adjustments. It is believed that a better health and longer life expectancy will result from good health due to maintaining a proper balance between ROS generation and antioxidant defenses against oxidative damages.

Conclusion

In conclusion, oxidative stress is an important contributor to the emergence and development of numerous diseases and disease processes affecting the vascular system, the brain, the immune system, and through the promotion of genetic mutations and increased infections in cancer progression. The cellular response to oxidative stress contributes to cellular damage, inflammation, and impaired tissue function of tissues exposed to increasing oxidative stress from the imbalance on the generation of reactive oxygen species (ROS) and the antioxidant defenses of the human body. The body does maintain a natural antioxidant mechanism to manage and minimize the damage caused by ROS, however, numerous environmental exposures, radiation exposures, and lifestyle choices can overwhelm this beneficial aspect of antioxidant action. Preventive measures can also help alleviate oxidative stress in humans and include implementing antioxidant-rich diets, regular physical activity and exercise, managing and regulating geneotypes, and minimizing exposures that increase/ amplify oxidative stress-induced damage. While ROS production must remain an ongoing physiologic process, further oxidative-stress induced dysregulation leading to injury and damage can be either prevented or no more than mitigated as long as the interaction between ROS production and the antioxidant or biological basis remains balanced from heightened to low levels within the body. Otherwise, a minimal increase in oxidative stress beyond anticipated oxidative burdens can lead to injury and damage of tissues through higher amounts of ROS production beyond its natural responses for bodily functioning through the metabolic process. Overall, it remains a goal to minimize the burden of oxidative stress in order to maximize health, retention of physical, cellular, and molecular function, longevity, and performance of the human body.

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