

# A Review On: “Oxidative Stress: Harms and Benefits for Human Health”.

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**ABSTRACT:** Oxidative stress is caused by a mismatch between the systemic manifestation of reactive oxygen species and a biological system's ability to quickly detoxify the reactive intermediates or repair the resulting damage. An imbalance between the synthesis and accumulation of oxygen reactive species (ORS) in cells and tissues, as well as the biological system's ability to detoxify these reactive products, causes oxidative stress. ORS can and do play several physiological roles, and they are normally produced as byproducts of oxygen metabolism; however, environmental stressors (i.e., UV, ionizing radiations, pollutants, heavy metals) and antitumor drugs contribute to greatly increased ORS production, causing an imbalance that leads to cell and tissue damage (oxidative stress). Several antioxidants, such as vitamin E, flavonoids, and polyphenols, have been studied in recent years for their actual or alleged anti-oxidative stress properties. While we often describe oxidative stress as harmful to the human body, it is also true that it is used as a therapeutic approach to treat clinical conditions such as cancer, with varying degrees of success. In this review paper, I'll discuss the most recent findings in the field of oxidative stress, emphasizing both its negative and positive effects on human health.

**KEYWORDS:** Oxygen Reactive species (ORS), manifestation, antitumor drug, Flavonoids, polyphenols, cancer.

## I. INTRODUCTION:

The concept of oxidative stress is prevalent in the chemical, biological, biochemical, and clinical-medical literature. The seemingly simple concept of an imbalance between oxidants and antioxidants, potentially leading to molecular damage, has expanded in recent years to focus on biological reactions, including redox signaling and control disruption. There are definitions for terms like dietary oxidative stress, postprandial oxidative stress, physiological oxidative stress, photooxidative stress, radiation-induced oxidative

stress, oxidant stress, pro-oxidant stress, and oxidative stress status. Reductive and nitrative stress are also addressed.

Oxidative stress has been linked to a variety of disease processes. Because the burden of disease in individuals increases with age, many scientists believe that oxidative stress plays a role in the ageing process and some disorders linked with old age, such as Alzheimer's disease. Numerous studies have found a direct link between ageing and oxidative stress. This chapter discusses the role of oxidative stress in ageing, as well as the relationship between oxidative stress and various neurodegenerative diseases that are more common in older people. A well-balanced diet rich in fruits and vegetables, frequent exercise, and a healthy lifestyle are the best defenses against oxidative stress, as addressed throughout the book. Antioxidant vitamins and supplements provide no additional benefit to healthy people. In fact, such supplements may be more harmful than beneficial. Antioxidant vitamins and supplements should only be taken under medical supervision.

### 1) Oxidants and Free Radical Production:

Nonenzymatic reactions, such as when oxygen reacts with organic compounds or when cells are exposed to ionizing radiation, can also be responsible for free radical production. During mitochondrial respiration, nonenzymatic free radical production can also occur.

Endogenous and exogenous sources of free radicals are both present. Endogenous free radical production is caused by immune cell activation, inflammation, ischemia, infection, cancer, excessive exercise, mental stress, and ageing. Exogenous free radical production can be caused by environmental pollutants, heavy metals (Cd, Hg, Pb, Fe, and As), certain drugs (cyclosporine, tacrolimus, gentamycin, and bleomycin), cigarette smoke, alcohol, and radiations, as well as chemical solvents and cooking (smoked meat, used oil, and fat). When exogenous compounds enter the body, they are

degraded or metabolized, and free radicals are produced as byproducts.

## 2) Pathophysiological Activities of Free Radicals:

It is a critical cell-to-cell messenger that is required for proper blood flow modulation, is involved in thrombosis, and is essential for normal neural activity. NO is also involved in nonspecific host defense, which is necessary for the elimination of intracellular pathogens and tumor cells. The induction of a mitogenic response is another physiological activity of free radicals. To summarize, free radicals are critical to human health when kept at low or moderate levels.

## 3) Detrimental Effects of Free Radicals on Human Health:

As previously stated, excess free radicals and oxidants cause oxidative stress; this is a harmful process that can negatively affect several cellular structures, including membranes, lipids, proteins, lipoproteins, and deoxyribonucleic acid (DNA). When there is an imbalance between the generation of free radicals and the ability of cells to remove them, oxidative stress arises. Excess hydroxyl radical and peroxynitrite, for example, can cause lipid peroxidation, causing cell membranes and lipoproteins to be damaged. This, in turn, will result in the formation of malondialdehyde (MDA) and conjugated diene compounds, both of which are known to be cytotoxic and mutagenic. If not strictly controlled, oxidative stress can cause the onset of a variety of diseases, both chronic and degenerative, as well as accelerate the ageing process and cause acute pathologies (i.e., trauma and stroke). Cancer and Oxidative Stress

- Cardiovascular Disease and Oxidative Stress
- Neurological Disease and Oxidative Stress
- Respiratory Disease and Oxidative Stress
- Rheumatoid Arthritis and Oxidative Stress
- Kidney Diseases and Oxidative Stress
- Sexual Maturation and Oxidative Stress

## 4). Exogenous Antioxidants and Human Health:

To counteract the effects of free radicals and oxidative stress, the human body employs a number of strategies based on enzymatic (e.g., SOD, CAT) and nonenzymatic (e.g., lipoic acid, glutathione, -arginine, and coenzyme Q10) antioxidant molecules, all of which are endogenous antioxidants. Aside from these, there are a number of exogenous antioxidant molecules of animal or vegetal origin that are primarily introduced through diet or nutritional supplementation.

In this section, we will go over the most important nutritional antioxidants and their effects on human health.

- Flavonoids
- Vitamin E

## 5) Prooxidant Agents in Therapy:

Prooxidant agents, in addition to their well-known negative effects on human health, have been studied and, in some cases, used as therapeutic agents, primarily against cancer diseases.

In this section, we will discuss two emerging prooxidant compounds with intriguing pharmacological activities, ascorbic acid (AA) and polyphenols, as well as the most well-known and widely used prooxidant in therapy, ionizing radiation.

### • Ascorbic Acid:

Ascorbic acid (vitamin C) is a water-soluble compound that belongs to the category of natural antioxidants. Ascorbate reacts with ROS, quenching them and promoting their conversion into semihydroascorbate radical, a chemical species with a low reactivity, effectively lowering the risk of cancer by suppressing free radicals and oxidative stress.

### • Polyphenols:

Under conditions such as high concentrations, high pH, and the presence of redox-active metals, phenolic compounds can exhibit prooxidant behavior, which is primarily based on the formation of an oxyl radical or a labile complex with a metal cation with redox activity. The formation of O<sub>2</sub> or a ternary compound between DNA, copper, and flavonoids can be caused by an oxyl radical. Caffeic acid, ferulic acid, and apigenin are polyphenols that accelerate oxidation by boosting intracellular synthesis.

Although polyphenols demonstrated the pharmacological potential to inhibit tumorigenesis and arrest cancer cell proliferation in animal models, the role of ROS generation remains poorly understood, owing to the fact that the vast majority of in vivo studies are limited to cancer growth arrest and apoptosis evaluation, and rarely, if at all, go deeper in the mechanistic explanation of a potential prooxidant action in vivo.

### • Radiation Therapy:

Ionizing radiation's ability to inhibit cancer cell proliferation is well understood and widely used in clinical practice. Over the last few decades, there has been a concerted effort to

comprehend the physical and molecular cellular responses that occur following ionizing radiation exposure. It is well understood that the most severe type of damage caused by this prooxidant physical agent is damage to DNA caused by the generation of radicals that indirectly cause DNA double-strand breaks (DSBs). These lesions are quickly repaired as a result of the rapid activation of the DSB damage repair mechanism, most notably nonhomologous end joining or homologous recombination, and the execution of a complex and finely tuned sequelae of the DNA damage response cellular signaling pathways (DDR). These responses range from protein posttranslational modifications and/or differential gene expression to the initiation of cell cycle reprogramming (e.g., radiation-induced arrest).

### Etiology:

Chronic diseases, such as osteoporosis, are caused by oxidative stress caused by reactive oxygen species (ROS).

1 There is ample evidence that oxidative stress causes bone loss, as revealed by epidemiologic studies.

2 and, as a result, it is a risk factor for osteoporosis. This chapter provides an overview of osteoporosis, the role of oxidative stress in bone cells – osteoclasts and osteoblasts – oxidative stress as a risk factor in the development of osteoporosis, and a review of studies on the use of antioxidants in osteoporosis prevention.



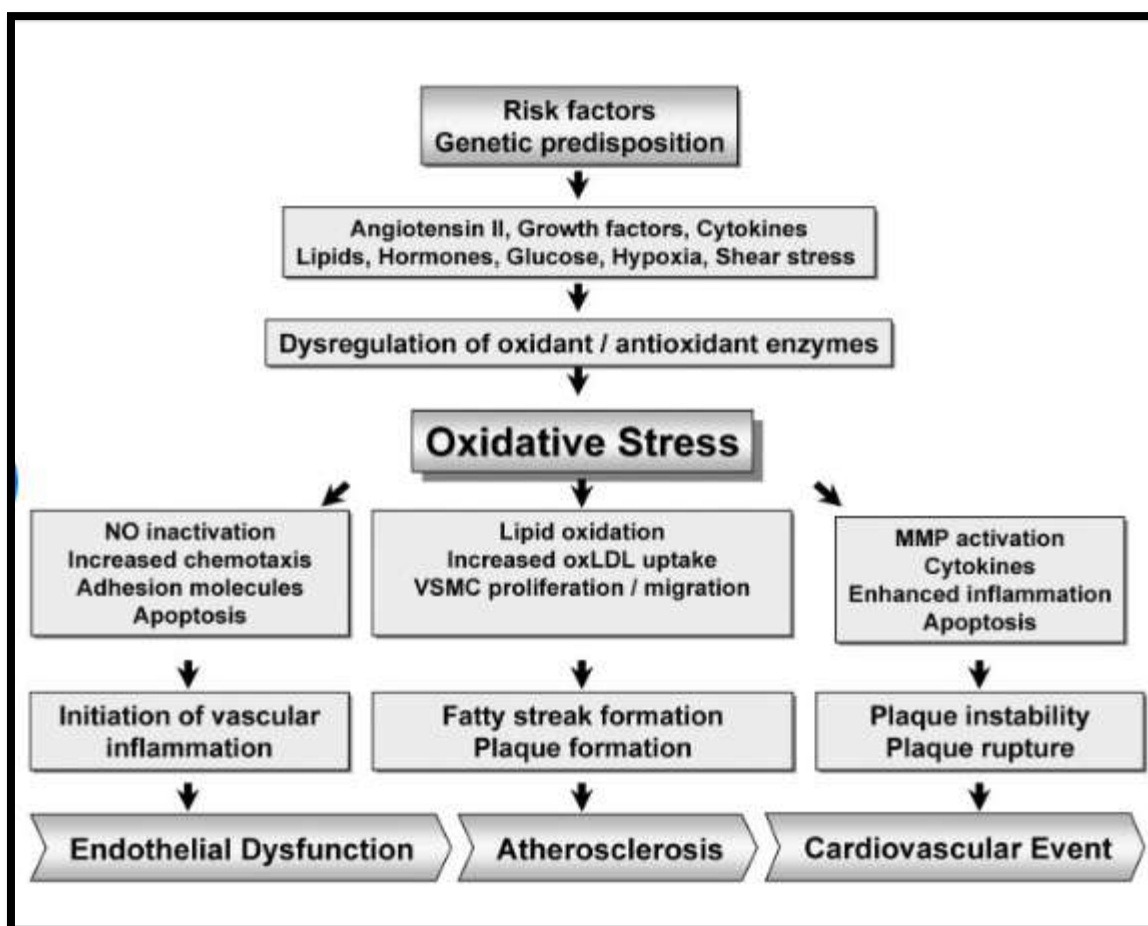
### Pathophysiology:

One of the most common causes of toxicity is oxidative stress. Chemical, physical, and microbial agents can all cause oxidative-mediated stress in tissues and cells. Furthermore, oxidative-mediated reactions are involved in a wide range of fundamental life processes, including cell respiration (mitochondria), lipid synthesis, metal metabolism, lysosomes, phagocytosis of foreign

bodies (immunity and inflammation), and xenobiotic biotransformation of organic compounds. To protect the integrity of cells or tissues, a suite of antioxidant compounds (reduced thiols, vitamins C and E, and catecholamines) and enzyme systems (superoxide dismutase, peroxidase, catalase, and glutathione reductase) are used to neutralize the release of reactive oxygen

species during these processes. Toxic agents may jeopardize the delicate balance between antioxidants and the production of reactive oxygen species. For example, the release of inflammation

precursors (hydroxyl anion, superoxide anion) during phagocytosis of invading particles/microbes may exceed antioxidant-protective mechanisms, resulting in oxidative (inflammatory) damage.



as DNA, protein, or lipid oxidation. Methods for measuring enzymes involved in the production of superoxide radicals (superoxide dismutase) and the elimination of hydrogen peroxide (peroxidase and catalase), total antioxidant capacity, and lipid peroxidation are demonstrated in this chapter. Age-related pigments and lipofuscins, which are oxidized insoluble fractions of lipids, proteins, and persistent oxidative stress, such as lipid peroxidation, could also produce age-related pigments and lipofuscins, may also result in the formation of age-related pigments and lipofuscins, which are oxidized insoluble fractions of lipids, proteins, and carbohydrates. Biomarkers of oxidative stress are one of the most important mechanisms by which chemical, physical, and biological agents contribute

to the emergence of a wide range of pathophysiological conditions.

Age-related disease is linked to oxidative stress: Several Age-Related Diseases are Associated with Oxidative Stress.

Age-Related Neurodegenerative Diseases

- Alzheimer's disease
- Amyotrophic lateral sclerosis
- Huntington disease
- Parkinson's disease

Age-Related Ophthalmological Disorders

- Glaucoma
- Cataract formation
- Macular degeneration

Age-Related Hearing Disorder

- Presbycusis

Age-Related Musculoskeletal System Diseases

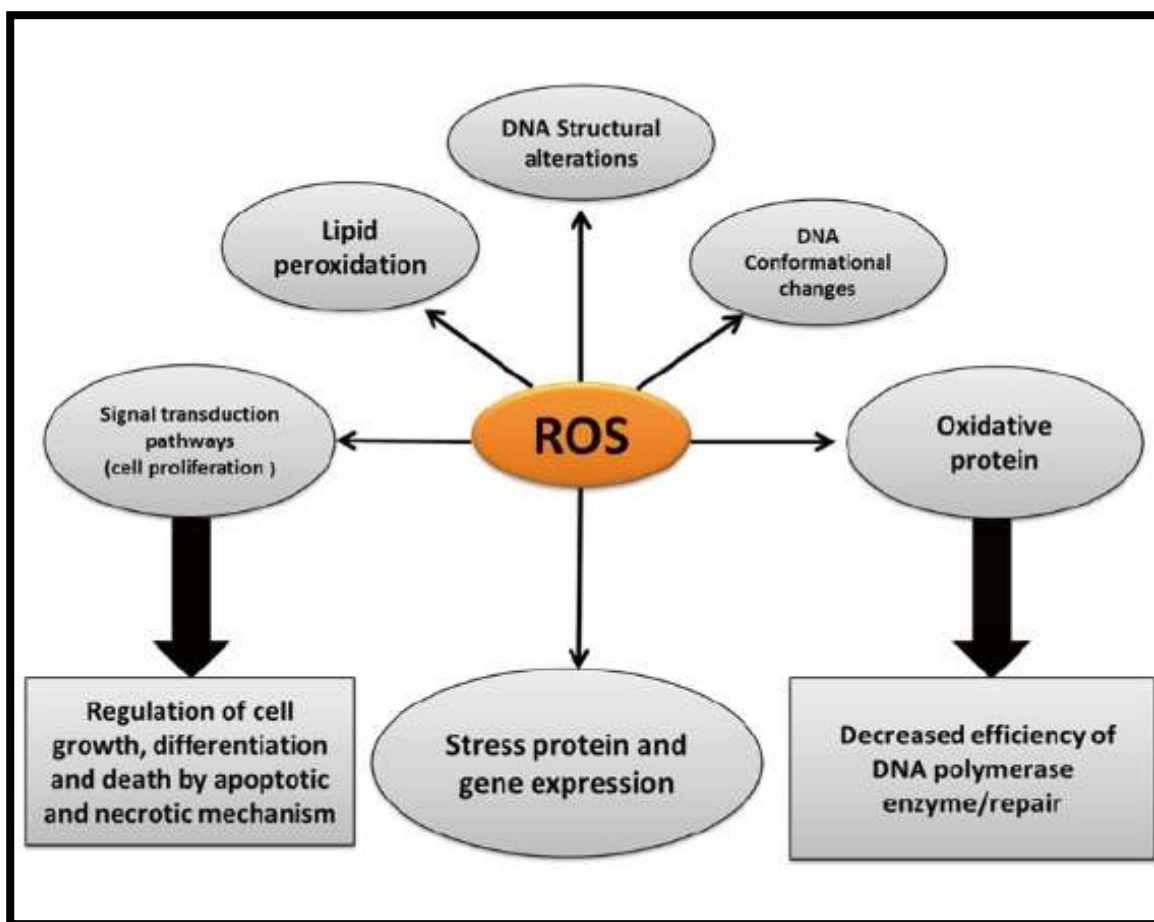
- Osteoarthritis
- Sarcopenia

Age-Related Skin Disorders

- Common skin lesions (dry skin, freckling, senile purpura, lentiginos, atrophicus, etc.)
- Benign tumor
- Premalignant tumor

- Malignant tumor (basal cell carcinoma, squamous cell carcinoma, malignant melanoma, etc.)
- Infectious disease (e.g., dermatophytosis)
- Autoimmune disease (e.g., contact dermatitis)
- Pressure ulcers, lower extremity ulcer
- Pruritus

Adverse effects of Oxidative Stress:



Environmental pollutants, chemotherapy and other chemicals, smoke, toxins, radiation, and diseases that can have a negative impact on fertility are all examples of oxidative stress. Cauda epididymal spermatozoa had low motility and high DNA oxidation levels at all times, according to our findings.

Causes of Oxidative Stress:

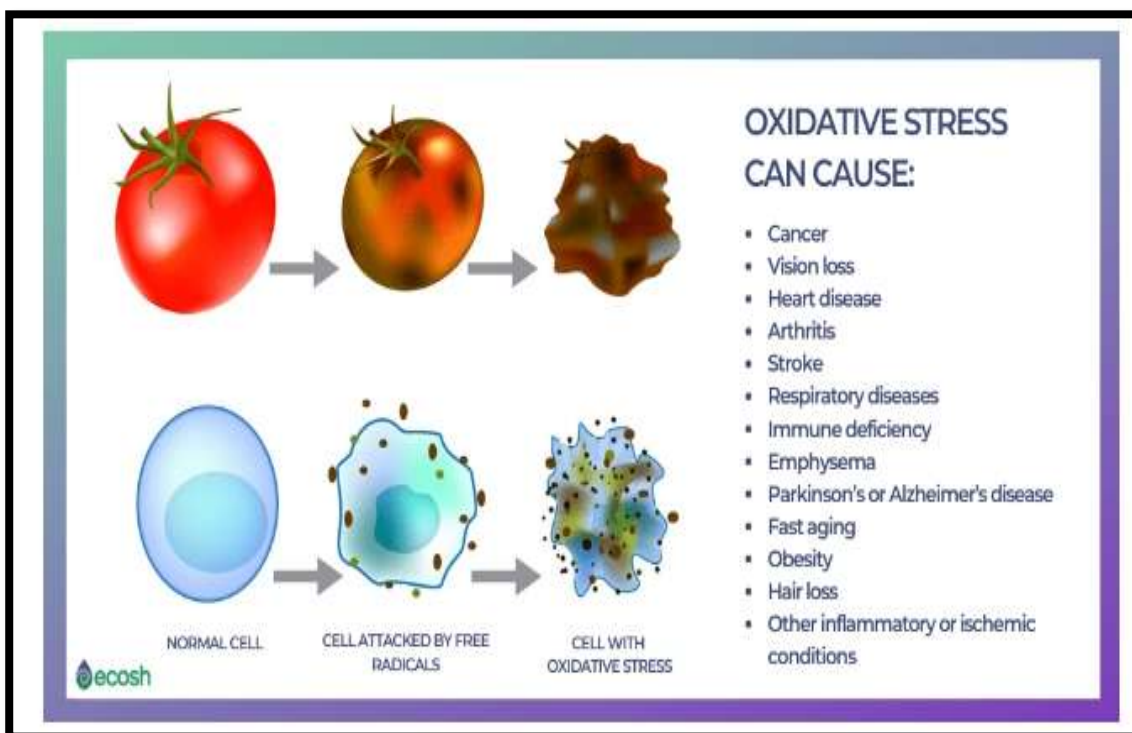
- Consumption of certain foods, particularly fried, refined, and processed foods, trans fats,

artificial sweeteners, carcinogen-containing foods, and certain dyes and additives.

- Excessive physical activity.
- Smoking.
- UV rays and cigarette smoke
- Toxins and pollution in the environment
- Radiation.
- Chemical exposure, such as pesticides and drugs, including chemotherapy, as well as the use of skincare products containing high levels of parabens and other chemicals.
- Industrial solvents.

- Alcohol.
- Some medications.

- In addition, low levels of antioxidants in food.



### Managing and preventing oxidative stress:

It is impossible to avoid free radicals and oxidative stress entirely. There are, however, things you can do to reduce the effects of oxidative stress on your body. The most important thing you can do is boost your antioxidant levels while lowering your free radical production.

One way to prevent oxidative stress is to make sure you're getting enough antioxidants in your diet. Eating five servings of fruits and vegetables per day is the best way to give your body what it needs to produce antioxidants. Fruits and vegetables include the following:

Broccoli, Carrots, Tomatoes, Olives, Berries, Cherries, Citrus Fruits, Prunes, Dark Leafy Greens, Berries, Cherries, Citrus Fruits, Prunes, Dark Leafy Greens, Broccoli, Carrots, Tomatoes, Olives

Other sources of antioxidants in the diet include:

Fish and nuts, Vitamin E, Vitamin C, Turmeric, Green tea, Melatonin, Onion, Garlic, Cinnamon

Other healthy lifestyle choices can also help to prevent or mitigate oxidative stress. Consider the following lifestyle options:

A routine of moderate exercise on a regular basis. This has been linked to higher natural antioxidant levels and less oxidative stress damage. Regular exercise has been linked to a longer lifespan, fewer ageing effects, and a lower risk of cancer and disease.

Don't light up. Avoid inhaling secondhand smoke as well.

When handling chemicals, exercise extreme caution. Avoiding needless radiation exposure, cleaning chemicals, and being aware of other forms of chemical exposure, such as pesticides used on food or in gardening, are all examples of this.

Be mindful of the environment. Carpooling and other environmentally friendly initiatives can help you and your community reduce free radical production.

Put on sunscreen. Sunscreen shields your skin from the sun's damaging UV rays.

Reduce your alcohol consumption.

Get lots of rest. A good night's sleep is critical for maintaining balance in all of your body's systems. Sleep has an impact on brain function, hormone production, antioxidant and free radical balance, and a variety of other things.

Overeating should be avoided. Overeating and continuous eating have been found in studies to

cause oxidative stress in the body more frequently than eating at regular intervals and in modest or moderate meals.

## II. CONCLUSION:

Free radicals and oxidative stress are both known to be harmful to human health. A large number of studies show that free radicals play a role in the initiation and progression of a variety of pathologies, ranging from CVD to cancer.

Antioxidants, as a class of compounds capable of counteracting oxidative stress and mitigating its effects on individual health, have piqued the interest of the biomedical research community not only because of their efficacy in disease prevention and/or treatment, but also because there is a widespread belief that they are free of significant side effects. If antioxidants can be very useful in preventing, managing, or treating human pathologies, it is also true that they are not immune to producing negative effects. We can conclude that oxidative stress, as a phenomenon, while being one of the major threats to people's wellness and health, can also be used as a treatment tool if and when we are able to fine-tune this process within the human organism.

## REFERENCE:

- [1]. Gabriele Pizzino, Natasha Irrera, [...], and Alessandra Bitto
- [2]. Piccoli C, Quarato G, D'Aprile A, Montemurno E, Scrima R, Ripoli M, Gomasaschi M, Cirillo P, Boffoli D, Calabresi L, Gesualdo L, Capitanio N. Native LDL-induced oxidative stress in human proximal 1 tubular cells: multiple players involved. *J Cell Mol Med* 2009 (in press).
- [3]. Droge W. Free radicals in the physiological control of cell function. *Physiological Reviews*. 2002; 82:47–95
- [4]. Young I, Woodside J. Antioxidants in health and disease. *Journal of Clinical Pathology*. 2001; 54:176–186.
- [5]. Medically reviewed by Timothy J. Legg, Ph.D., CRNP — Written by Megan Dix, RN, BSN — Updated on September 29, 2018
- [6]. Ilaria Liguori, Gennaro Russo, [...], and Pasquale Abete
- [7]. Kimura S, Zhang GX, Nishiyama A, Shokoji T, Yao L, Fan YY, Rahman M, Abe Y. Mitochondria-derived reactive oxygen species and vascular MAP kinases: comparison of angiotensin II and diazoxide. *Hypertension* 2005; 45: 438–444. CAS Goog
- [8]. Pei You Wu, Eleonora Scarlata, Cristian O'Flaherty, Department of Surgery (Urology Division), McGill University, Montréal, QC H4A 3J1, Canada. Department of Pharmacology and Therapeutics, McGill University, Montréal, QC H3G 1Y6, Canada
- [9]. Rodrigo R, Rivera G. Renal damage mediated by oxidative stress: a hypothesis of protective effects of red wine. *Free Radic Biol Med* 2002; 33: 409–422.
- [10]. Guarnieri G, Zanetti M, Vinci P, Cattin MR, Pirulli A, Barazzoni R. Metabolic syndrome and chronic kidney disease. *J Ren Nutr* 2010; 20: 19–23.
- [11]. Pizzino, Natasha Irrera, [...], and Alessandra Bitto
- [12]. reviewed by Timothy J. Legg, Ph.D., CRNP — Written by Megan Dix, RN, BSN — Updated on September 29, 2018
- [13]. Yoshioka J, Schreiter ER, Lee RT. Role of thioredoxin in cell growth through interactions with signaling molecules. *Antioxid Redox Signal* 2006; 8: 2143–2145. CAS Google Scholar